DISEASES OF ANIMALS IN SOUTH AFRICA

C. R. EDMONDS

Patricia H. Moore.

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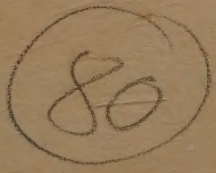

From: N.S.

DISEASES OF ANIMALS IN SOUTH AFRICA

I expect to pass through this world but once. Any good, therefore, that I can do, or any kindness that I can show to any fellow-creature, let me do it now. Let me not defer or neglect it, for I shall not pass this way again.—AUTHOR UNKNOWN.

DISEASES OF ANIMALS IN SOUTH AFRICA

BY

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PREFACE

THE cause that led to the attempt of this work was the feeling that there was a need for a book dealing with the more serious diseases affecting farm animals in South Africa as distinct from other lands.

The admirable works dealing with animal diseases in other parts of the world, and to which we in South Africa are greatly indebted, do not treat the diseases peculiar to this sub-continent in the manner, if at all, in which they have been chronicled from actual experience in this country by a number of veterinary surgeons and other writers in our agricultural press.

To such writers I am indebted for much information, also to the *Rhodesia Agricultural Journal*, for many of the plates, and to L. E. W. Bevan, Esq., Veterinary Bacteriologist, for photographs that are reproduced herein.

My reference library, though limited in numbers, has been of great assistance, and I beg to tender my thanks and appreciation to the authors of "A System of Veterinary Medicine" (E. Wallis Hoare), "Special Pathology and Therapeutics of the Diseases of Domestic Animals" (Hutyra and Marek), and "Neumann's Parasites" (MacQueen) for the help derived from consultation of their volumes.

I have endeavoured to avoid as many technicalities as possible, and in this and many other ways I have been greatly assisted by C. E. Gray, Esq., M.R.C.V.S., Principal Veterinary Officer of the Union of South Africa, who kindly read through and made additions to the manuscript.

Into the history of South Africa the experiences of the pioneers and Voortrekkers enter greatly, but many of them are at the present time unknown, while others have left behind them records

of their travels over the unknown seas and land. Such records may at the present day be considered very crude, or the spoor over the veld may be long and winding, but each record served its purpose of warning the oncoming generations of rocks and other dangers ahead and advising the best-known means of the time to avoid them.

The information so conveyed has been added to and improved upon. If this effort of mine serves a similar purpose and meets a like fate, I shall feel repaid for the hours the writing has occupied.

C. R. EDMONDS.

BULAWAYO.
November, 1921

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DISEASES OF ANIMALS IN SOUTH AFRICA

INTRODUCTORY

CLASSIFICATION OF PARASITES

" Great fleas have little fleas upon their backs to bite 'em,
And little fleas have lesser fleas, and so *ad infinitum.*
And the great fleas themselves in turn have greater fleas to go on;
While these, again, have greater still, and greater still, and so on."

SCIENCE daily increases the knowledge and adds to the number of diseases that are discovered to be caused by inferior organisms; so much so, that research in both human and veterinary pathology at the present time is greatly directed to the organismal cause of disease. Needless to say, our present knowledge is incomplete, but as I think the correct way to study the more serious diseases is by a grouping of the organisms causing them, I have attempted to classify them accordingly. No doubt my classification will be found sooner or later in some or many respects to be incorrect, and I realise there are objections to this method of classification; but, after all, it is only classification which does not affect the nature or description of any particular disease.

When a living organism takes up its abode temporarily or permanently on or within another living organism, the condition is spoken of as symbiosis.

There are three degrees of this union recognised:

Mutualism, in which both the organisms are benefited.
Commensalism, in which one profits whilst the other is unharmed.
Parasitism, in which one profits and the other suffers injury in some way.

When the relations between size and strength are such that the profiteer is inferior to its victim, the former becomes the parasite; and when it draws all its subsistence from the latter, the victim is then known as its host or carrier.

The parasites which live on the domestic animals (mammals and birds) belong to the vegetable and animal kingdoms.

Vegetable Parasites.—The majority of these are included in the groups of Schizomycetes and Bacteria. They are formed of a single cell, destitute of a nucleus, and are the cause of many diseases the study of which is known as Bacteriology.

In bacteriology the organisms are classed as follows:

1. PATHOGENIC (disease-producing).

2. SAPROPHYTIC (or non-pathogenic).—These cannot live in animal tissue, and would maintain their existence if every animal were dead. They are found in the greater majority in nature.

The pathogenic organisms are divided into two classes:

A. *Obligatory Parasites.*—Includes all those which are incapable of multiplying outside the animal body. They cause infectious and contagious diseases—amongst others, tuberculosis, glanders, pleuro-pneumonia, contagious abortion, etc.

B. *Facultative Parasites.*—Includes all those which can maintain their existence as saprophytes or as parasites. They are only accidentally the cause of disease. Diseases caused by them may be sporadic—all are sometimes so—and the great majority are neither contagious nor infectious. Tetanus is a good example of this class of disease. Not 1 per cent. of the organisms existing ever cause this disease, and the organism would live if all animals died. Other diseases caused by this class of organism are quarter evil, actinomycosis, necrosis bacillus, swine erysipelas (red soldier), *Staphylococcus pyogenes aureus*, *Streptococcus pyogenes*.

It is believed there is every possibility that certain organisms—anthrax and fowl cholera, for instance—whilst being entirely obligatory parasites in cold climates, may exist as facultative organisms in hot climates.

In some cases the actual organism that is the real cause of the disease has not yet been determined; for instance, catarrhal roup and diphtheritic roup in poultry.

Animal Parasites.—Three sections only of the animal kingdom contain parasites of the domesticated animals; these are the Protozoa, Worms, and Arthropodes.

1. THE PROTOZOA.—The immense majority are entirely microscopic, but a few are visible to the naked eye, although only composed of a single cell.

They are divided into four classes:

(1) *Rhizopoda.*—This includes the order Amœbina, genus amœba. Amœba contains a pathogenic species for man, but it is doubtful if any are pathogenic for the lower animals.

(2) *Mastigophora.*—This class is also known as the Flagellata, and contains the Trypanosomes.

(3) *Sporozoa*.—The animals of this class are, without exception, parasites. There are no free-living ones. This class is divided into five orders:

A. *Gregarinida*.—This order does not include any species of pathological importance. They are exceedingly common in the very low animals.

B. *Coccidiidea*.—This includes the Coccidea, which are seriously pathogenic for many different species of animals.

C. *Hæmosporidia*.—This order includes the malarial parasites and the plasmodia.

D. *Nyosporidia*.—This order is of no importance to medical men or veterinary surgeons.

E. *Sarcosporidia*.—This order contains species parasitic in man and in the lower animals. The so-called Meischer's tubes belong to this order.

(4) *Infusoria*.—Contains no species of pathological interest.

2. The Worms.—This section contains a vast collection which do not form a very natural zoological class. It contains:

(1) Hirudines, the leeches, and Helminthes.

(2) Helminthes are divided into two classes:

A. Platyhelminthes, with bodies generally flat. These are divided into three orders:

(i.) Cestoda, which lead a parasitic life.
(ii.) Trematoda, which lead a parasitic life.
(iii.) Turbellaria, non-parasitic.

B. Nemathelminthes, or round-worms, are divided into two orders:

(i.) Acanthocephala.
(ii.) Nematoda.

3. The Arthropoda.—These are divided into four classes: Crustacea, Arachnida, Myriapoda, and Insecta. The following two are the only ones which contain species parasitic in the domestic animals:

(1) *Arachnida* are air-breathing, head and thorax usually fixed together, four pairs of feet, and no wings.

(2) *The Insecta* are air-breathing, possessing a windpipe, body divided into three distinct parts (head, thorax, and abdomen), three pairs of legs, and, most frequently, two pairs of wings. There are many orders, only two of which—Diptera and Hemiptera—contain parasitic species.

NOTIFIABLE DISEASES

THE laws of South Africa, in common with those of other countries, place certain obligations upon the owners of stock that are, or may be, suffering from certain diseases, such as prohibiting the owner from removing, or allowing such stock to be removed, from his land, and compelling him to report the existence, or suspected existence, of such disease to an official appointed to receive such reports.

In the Union of South Africa, these obligations are set out in Act No. 14 of 1911. In Southern Rhodesia they are detailed in Ordinance No. 9 of 1904, known as the Animals' Diseases Consolidation Ordinance. These two enactments are very similar in the conditions laid down, but for the purpose of this book it is only necessary to table the diseases of which notification must be made. These are as follows:

In the Union of South Africa and Southern Rhodesia.

Anthrax.
East Coast fever.
Epizootic lymphangitis.
Foot and mouth disease.
Glanders and farcy.
Mange in equines.
Pleuro-pneumonia (lung-sickness).
Rabies.
Rinderpest.
Scab in sheep and goats.
Swine erysipelas.
Swine fever.
Tuberculosis.

In the Union of South Africa only.

Sheep pox.
Trypanosomiasis.
Ulcerative lymphangitis.

In Southern Rhodesia only.

Contagious abortion.
Heartwater.
Redwater.
Quarter evil or sponszeikte.

It must be understood that the foregoing comprehensive list includes diseases that have never existed in South Africa, but were probably included in anticipation of their appearance; also others that have been entirely eradicated in some cases, and nearly so in other cases, as well as diseases, such as redwater and quarter evil (Rhodesia), that can hardly lay claim to be regarded as notifiable diseases.

Predilection Seat.

In many bacterial diseases the position of the lesions is due to the proximity of the point at which the bacteria penetrated the body of the animal, or to vascular or lymphatic connection between the different parts. This is obviously so in cases of actinomycosis, and is for the most part so in tuberculosis; but there are other instances in which it is quite impossible to explain the reason on anatomical grounds. Foot and mouth disease is perhaps the best example in which the position of the lesions is entirely separate from the point of penetration of the organism. This can be proved experimentally by producing the disease by inoculation, by ingestion, or other method, and the lesions always appear in the same parts.

It is a known fact that in the early stages of foot and mouth disease the virus is in circulation in the blood. This can be proved by inoculation. At a later stage it is only at particular places where it multiplies. These places become the seat of the lesions, and are called predilection seats. It is not possible to explain why certain places are predilection seats for different diseases.

Koch's Postulates.

Fairly early in the history of bacteriology, Professor R. Koch laid down certain conditions that he contended must be fulfilled before a bacterium could be definitely held to be the cause of a disease. These conditions are known as "Koch's postulates," and are as follows:

1. The bacterium must be constantly present in the disease, and not present in animals free from the disease.

2. The bacterium must be obtained in a state of purity, and be cultivated through several generations outside the animal body.

3. With such a culture one must be able to experimentally produce the disease.

4. From the experimentally infected animal one must recover the organism and identify it.

Disposal of Carcases.

The effective disposal of the carcases of animals, whether they have died from disease or from other causes, is of particular importance to the South African stockowner.

In addition to the risk of disease being spread by diseased carcases that have not been effectively disposed of, there is the possibility, as has been recently proved by experiments in connection with lamziekte, of very serious disease being set up through animals eating the bones and flesh of decomposing carcases. The question of the proper disposal of carcases of farm animals is complicated to a certain extent by the existence of the native races, who, in many cases, will eat meat of dead animals and animals killed on account of disease that a European under no circumstances would think of consuming. Further, numerous cases have been recorded of carcases buried under the instructions and supervision of a European in the day being dug up by the natives in the night, and the flesh taken away and consumed. The question of allowing natives to consume the carcase of any animal that dies is a doubtful practice, for the reason that in places where this practice has been followed many convictions have been obtained against natives for secretly injuring animals to such an extent as to cause death. One of the methods followed is to poke a sharp-pointed stick by way of the anus some distance into the animal's body. The inflammation and hæmorrhage caused by the stick piercing the bowels and other internal organs result in the death of the animal, the carcase of which the natives expect to fall to them. With smaller animals a hat pin may be forced direct into the heart. This leaves but small trace of its passage. Or a blow on the head with a knobkerrie leaves a bruise only in a position that may be easily overlooked.

Wild animals, too, will scratch up the carcases that have been buried, and there is no doubt that the most effective way of disposing of carcases is by cremation. In the parts of the country where wood is plentiful this can be done at little expense or trouble. In Rhodesia the method pursued, if simple, is very wasteful of fuel. Wood is piled around and on top of the carcase,

and set alight, about a waggon-load of wood being used. A method more economical of fuel is to dig a trench in the form of a cross, each of the four wings about 7 feet long, about 9 inches (the width of a shovel) wide, about the same depth at the intersection, becoming gradually shallower towards its extremity. The soil that is dug up is banked up in the angles formed by the intersection of the trenches, and on this, across the trench, iron rods or long rocks, if available, are placed; if not, big logs of wood, on which is placed the carcase, either intact or dismembered. If the latter, the parts should be interspersed with wood, the bowels and their contents being placed on the pile last. By the trench method less wood is required, the carcase is cremated in a shorter time, and a more thorough and workmanlike job results.

THE DETECTION OF DISEASE

It is a matter of the greatest importance to detect disease early. If the malady happens to be only an ordinary ailment, treatment applied in the first stages stands a better chance of being successful than if applied later on; and if the disease is a contagious one, early detection will facilitate the adoption of measures to prevent its spread.

Therefore it behoves an owner to get to know his stock as well as possible. The degree to which this can be carried out will depend upon the powers of observation of the owner, and the manner in which the particular stock are handled; for instance, animals that are stabled and under constant observation should be known thoroughly. Each animal's peculiarities will be understood, and any departure from the normal will be regarded with suspicion as denoting that something is wrong. Animals that run in paddocks day and night cannot be known so well individually, but to the experienced eye indications are not lacking directly there is anything amiss in such animals.

Symptoms of Ill-Health.

One of the first indications of ill-health amongst animals is a loss of appetite. With hand-fed animals this is quickly detected, with others the owner may receive the information by the herd reporting the fact, or he may find it out for himself by observing the "empty" appearance of the animal. Whilst being off its feed denotes that there is something wrong with the animal, it must not be taken for granted that because an animal keeps on feeding there cannot be much the matter with it, as this is not so. East Coast fever may be quoted as one disease in which the appetite remains good although the beast may be seriously ill; therefore, it comes about that the retention of an appetite in an animal that is obviously ill may aid in the diagnosis of the disease from which it is suffering.

Another early indication of ill-health is for the animal to isolate itself from its fellows. Of course, different animals different ways. Whilst a donkey may be frequently seen standing under

a tree apart from the others, a cow in a similar position would at once cause an investigation to discover the reason for this isolation. Likewise, if a troop of animals are grazing, but there is one lying down or standing apart, or if an animal is missed from its usual place in the troop when they are moving, and is seen to be lagging behind, it at once creates suspicion. Amongst animals that are accustomed to move or graze in troops, it will be found that certain animals are invariably in front leading and others at the back, and an animal out of its customary place is quickly noted by the experienced stockman.

Naturally, it is amongst the tail-end of the troop that animals in ill-health can be sought, and if an animal is seen to have a staring coat, to be tucked up, dull, weak, or lame, a closer examination of it will be made.

The Eye.—The eye is a good indicator of the state of health of the animal. In good health it is clear and bright, but assumes different appearances in different maladies. The glassy eye frequently denotes fever or pain, the eyes sunk deep in their sockets usually denotes serious illness. They may appear to be more sunken than they actually are owing to swelling of the eyelids. Then there is the protruding eye, as seen in cases of the dikkop form of horse-sickness. At times the eyeballs, owing to the pressure from the swelling behind them, protrude to a very great extent. Again, there is the wild, excitable-looking eye, denoting some nervous affection or poisoning. Frequently there is a discharge from the eyes. If it is only from one eye, it usually denotes local trouble to that particular eye, or ophthalmia. A discharge from both eyes is a symptom of certain diseases.

Mucous Membranes.—The mucous membrane—that is, the red membrane lining the inside of the eyelid, lips, and mouth—is one of the greatest helps in diagnosing disease. In health, it is a clear pink colour; a deep red is found in cases of inflammation. This deep red frequently takes on a leaden, dirty hue in cases where the blood is charged with organisms or their products, in some of the diseases affecting the blood. In other diseases blood-red spots are found on the mucous membrane of the eye. A pale or anæmic membrane denotes a loss of blood from hæmorrhage, disease that has destroyed the blood-corpuscles, or parasitism that has abstracted it. A yellow or jaundiced membrane is frequently present as a symptom of disease. In animals with white skins this colour will frequently be observed around the eyes, mouth, and ears. A discharge from the nostrils should also

be taken into consideration, and the moist muzzle in healthy cattle and the cold nose of the dog must not be forgotten.

Respiration.—The breathing of an animal may be little altered in many diseases; in others, it will be quickened or laboured. In animals in a comatose condition, it may be slower and sometimes snoring. The animal may be breathing with its chest more than usual, as seen by the ribs expanding and contracting greatly; on the other hand, the ribs may be held as firmly fixed as possible, and the muscles of the abdomen are prominently brought into play. Great use is made of these muscles in cases of horse-sickness.

The Pulse.—The pulse is, perhaps, more useful as an indicator of the seriousness of the case than as a diagnosis for any particular disease, with the exception of heart disease. It is felt in the horse at the inside of the lower jaw; in cattle, just at the outside of the jaw, or the under-surface of the tail; in the dog, pig, or sheep it can be felt on the inside of the forearm or thigh. To differentiate between the various pulses requires a considerable experience, and to be able to test whether the pulse is strong or weak is about as much as the average layman can do.

Temperature.—The temperature of the animal as registered by the clinical thermometer is most useful in many diseases. To shake the mercury down below the normal temperature mark of the animal, to spit on the thermometer, insert it in the rectum, and hold it there for two minutes, is very simple. So is the use of a rifle, but the efficient use of it depends upon the man behind it; and so with a thermometer. This is merely put in as a word of warning to novices not to rely too much on a thermometer. Horse-dealers are fond of using the expression, "What you can't see you won't feel." Likewise, in most animal ailments there are other symptoms as well as a rise of temperature present. A rise in temperature can, and frequently does, take place without there being anything seriously wrong with the animal; on the other hand, in a certain number of diseases a rise in temperature is the first indication of it.

There are many other general symptoms of ill-health in animals. The coat is often an indicator of something being amiss. In good health it has a glossy appearance; in disease it frequently becomes harsh and dry. This is what commonly occurs when an animal is the host of a number of worms (intestinal). The number of ticks on an animal is at times an indication of disease, or, perhaps, a condition of the skin preferred by the tick. This is seen when only one or two animals in a large troop are badly

tick-infested, and in such cases it is difficult to decide between cause and effect: whether the ticks cause the condition of the skin, or whether the condition of the skin attracts the ticks to the animal.

The coat, instead of having the sleek appearance seen in good health, may be "staring," as if the animal were cold. This condition is frequently observed in animals with fever. The external temperature of the body as revealed by feeling the extremities—the feet and legs, ears, horns, etc.—with the hand may show that they are cold, which indicates that the circulation of the blood is not as it should be, and that the heart is not doing its duty of forcing the blood throughout the body; or they may be hot, as in inflammation, or alternately hot and cold.

The skin, which is normally soft and pliable, and somewhat loosely attached to the body, may become hard and "hide-bound" in disease.

"Hanging of the ears," as it is usually called when the ears are allowed to droop, is one of the first indications of disease in many instances. To observe this symptom it is often necessary to approach the animal very stealthily, for if it is disturbed it may prick up the ears and appear quite normal in this respect. Lameness, as in the case of quarter evil, may be the first indication of the existence of disease.

With herbivorous animals in particular, the state of the digestive organs indicates to a great extent the state of the general health of the body. In ruminants the loss of rumination—that is, cessation of the process of chewing the cud—is an indication of ill-health. A depraved appetite, such as cattle chewing bones, or they and other animals eating dirt, is usually a sign of lack of something in the food, the digestive organs being out of order, or, perhaps, more serious trouble.

The discharges from the body also assist greatly in the detection of disease. The fæces should be examined to see if the food has been properly masticated and digested, or if it contains traces of any internal parasites. A small clot of blood or a piece of mucus on the dung tells a tale of some cause for its presence there, and the habit of the cattleman of frequently flattening out with his boot the droppings of cattle and looking at them must not be regarded as a mere habit similar to that of sucking a straw in the mouth; it has a real object behind it. The colour and consistency of fæces conveys a world of information to the experienced man.

The urine, too, has its own tale to tell. It may be scanty and

high-coloured, as it is in many febrile conditions; it may be profuse, as in diabetes; it may be discharged frequently, or with pain; it may contain a great amount of sediment; it may smell strongly of ammonia; it may be altered in colour, resembling blood in redwater, coffee-coloured in biliary fever, bile-stained in some affections of the liver, and in other ways. An examination of its nature will assist in the detection of the existence of disease and aid in correct diagnosis of the malady. But the only way that a correct interpretation of the signs of disease and the malady in particular they indicate can be made is by close observation extended over a sufficiently lengthy period. The length of time required and the ultimate ability attained depends, of course, upon the observer.

Smears.—Blood and other smears for microscopical examination in many cases, when examined by a bacteriologist, provide the

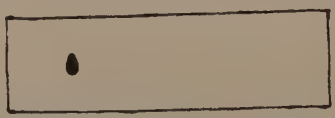


FIG. 1.—DROP OF BLOOD ON GLASS.

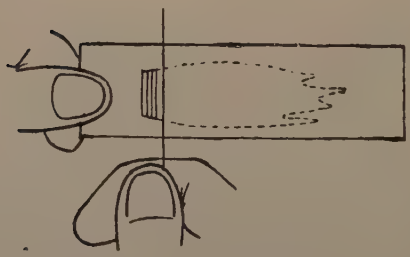


FIG. 2.—THE SAME SPREAD OUT INTO A THIN FILM.

final and absolute proof of the existence of certain diseases; therefore, it is well to know how to take them.

In the living animal a blood-smear may be obtained readily from the ear of the animal by clipping the hair off its edge, then, washing clean and drying afterwards, cut a slit in the margin of the ear with a knife or pair of scissors, and after a few drops have escaped catch one drop on the flat surface of the glass slide. Clotted blood will not make a good film for examination.

Smears may also be obtained in the living animal from swollen lymphatic glands and the spleen by plunging a hypodermic

needle into them and sucking the substance into the barrel by withdrawing the piston. Naturally, it is desirable first to clip and wash the skin, but the spleen operation perhaps entails a greater knowledge of anatomy than the average man possesses.

In the dead animal a smear from any organ is easily obtainable.

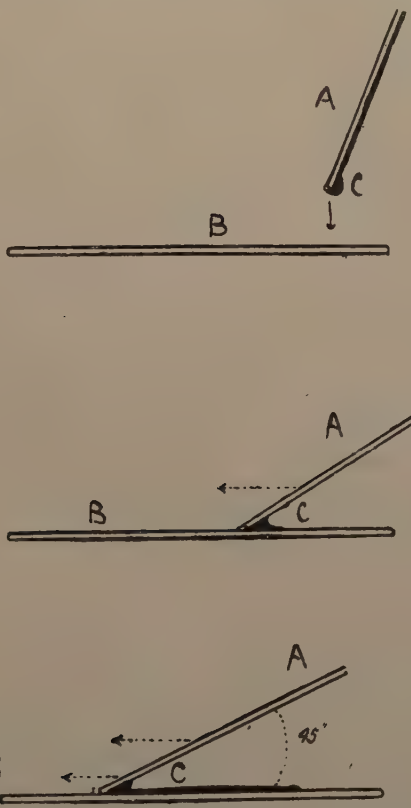


FIG. 3.—METHOD OF PREPARING A BLOOD SMEAR.
A, First slide; *B*, second slide; *C*, drop of blood.

A slide is a piece of thin flat glass 3 inches long by 1 inch wide with the edges rounded off. If unobtainable, a piece of window glass can be used. This should be thoroughly well cleaned, and afterwards only held between the fingers and thumb by the edges; the flat side should not be touched by the finger or thumb. The object in almost all smears is to get as thin a layer as possible on the glass, and this to be done without crushing the material

to be examined. Therefore, after a drop of blood has been placed on the slide about an inch from one end, the edge of a piece of tissue paper—a cigarette paper will do well—should be placed at the side of the drop, and after the blood has flown along the edge of the paper the latter should be gently drawn along towards the other end of the slide, which is then held in the sun until it is quite dry, when it should be wrapped in clean paper to prevent dust getting on it. Always take two or three smears; never rely on one only.

If tissue-paper is not obtainable, a needle used flat across the glass to draw the blood after it can be used, or even the edge of another slide in a similar manner.

Smears of spleen pulp and other smears are prepared in a similar way.

In taking smears for examination it is, of course, essential that smears should be of the substance in which the organism that is suspected is usually found. For instance, if the organism is confined to the lesions, it is no use taking a blood-smear from the ear. A study of the diseases will indicate where the organism is usually found, and where it is not otherwise stated, the material can be lightly spread across the slide in the manner indicated. With very watery materials it is advisable to place these on the slide in a slightly thicker layer, and to allow the excess moisture to evaporate, or heat the glass to cause it to do so. In taking a smear from a solid tissue, this can often be best done by simply dropping the slide upon it.

PART I

DISEASES CAUSED BY VEGETABLE PARASITES

PYOGENIC (PUS-FORMING) BACTERIAL DISEASES

Septicæmia.

This term was formerly used to denote a condition in which the blood was invaded by pus-forming organisms. At the present time a septicæmia denotes an invasion of the blood by any bacterium. For instance, anthrax is spoken of as a typical septicæmia.

The pus-forming bacteria are frequently responsible for producing this condition, but it is well to remember that when a septicæmia is spoken of, it does not necessarily mean that it is caused by one of these organisms.

Sapræmia

is commonly known as septic intoxication, and is caused by the absorption into the blood of toxins (poisons) produced by putrefactive bacteria. Good examples of this may frequently be seen in retention of the afterbirth in cows. In a septicæmia the blood contains bacteria, and is infective; in sapræmia it does not contain bacteria, and is not infective for other animals.

Pyæmia

denotes abscesses in various parts of the body due to infective pus-forming material or organisms being transported from one site to another by the lymph or blood-stream, and creating abscesses in fresh centres.

Common Pus-forming (Pyogenic) Bacteria.

These are the following staphylococci: *Staphylococcus pyogenes aureus*, *S. pyogenes albus*, *S. pyogenes citreus*, *S. cereus albus*, *S. cereus flavus*, and *Streptococcus pyogenes*.

Of the staphylococci, the last two named are not very

frequently found in pus; of the others, the first named is the commonest met with, the other two being, as a rule, found with it when they are present.

Staphylococcus Pyogenes Aureus.

This bacterium is responsible for a variety of diseases. Both in man and in the lower animals it is frequently responsible for:

1. Acute circumscribed abscesses in connection with, or independently of, wounds; boils in the human subject, and analogous lesions in the lower animals. In Rhodesia boils are frequently seen in yearling cattle, usually in the region of the groin and thighs. In the majority of cases one boil only forms. They do not show any tendency to recur or spread, as frequently happens in man.
2. Suppuration at the surface of wounds that are healing by granulation (open wounds).
3. Acute osteomyelitis (inflammation of the bone marrow, or of the bone and marrow).
4. Acute endocarditis (inflammation of the membrane lining the cavities of the heart).
5. Cases of pyæmia with multiple abscesses in the internal organs, especially in the cortex of the kidney.

Nature of the Organism.—The *Staphylococcus pyogenes aureus* is a facultative parasite almost ubiquitous in its distribution. It is present in ordinary water, dust, and dirt; also upon the skins of men and animals. The main method of multiplication is as a saprophyte.

It is, in the main, a tissue parasite, and confined to the lesions, but it may be transported by the blood or lymph stream. It is never present in the form of a septicæmia, and it is rare that one can detect it in the blood by microscopical examination.

Lesions occur in all the domesticated animals and man; no warm-blooded animal is immune to it. The natural method of infection is usually through a wound or abrasion, but probably these are not absolutely necessary, as experimental infection can be set up by rubbing the organism into the intact skin. The organism is not infective by ingestion.

Streptococcus Pyogenes

is a facultative parasite. In the animal body it is usually confined to the lesions. It causes pyæmia and endocarditis. In the small laboratory animals—especially in the mouse—the inoculated disease is frequently septicæmic in character.

It agrees closely with the common pyogenic staphylococci, but it is also responsible for a class of lesions that *S. pyogenes aureus* and *albus* are not often responsible for, as human erysipelas caused by the *Streptococcus erysipelatis*, which was once so named; but it has been found impossible to distinguish this by morphological, cultural, or staining reactions from the streptococcus found in acute cases, and it is now believed to be the *Streptococcus pyogenes*. Erysipelas is frequently contagious in the human subject, and it spreads as erysipelas, and not as a suppuration—that is, pours out a liquid exudate, not pus. Erysipelatous lesions are met with in the lower animals, but have started from a wound.

Streptococci are also a common cause of pleurisy, pericarditis, and peritonitis; and in the human subject and the lower animals are also often responsible for metritis (inflammation of the womb). Attacks of acute and subacute mastitis (inflammation of the udder) in the cow are frequently caused by streptococci.

STRANGLES

Synonyms.—Infectious Rhino-adenitis, Febris Pyogenica, Distemper in Horses. In South Africa it is commonly known as Nieuweziekte.

History.—The contagious nature of the disease was first pointed out by Solleysell in 1664, but, previous to that time, the disease had been described by Blundevill in 1609, and by Markham in 1623. In 1790, Laforse proved the contagious nature of the disease by inoculation. Similar experiments were subsequently made, and confirmed, by a number of investigators. In 1888, Schutz discovered and described the organism which is now regarded by most authorities as the cause of the disease—viz., the *Streptococcus equi* or the streptococcus of Schutz.

Nature.—The organism causing strangles represents a distinct species endowed with pathogenic powers not possessed by any of the streptococci found in man. The question of whether the parasite is obligatory or facultative must be answered by how one decides as to whether the disease is purely a contagious or a sporadic one. Experience points to strangles being a purely contagious disease, and the parasite an obligatory one. The organism is confined to the lesions and discharges in ordinary cases, and to the upper air passages. The disease may be pyæmic in character, but it is not septicæmic. It is confined to the horse family. Mules are less frequently attacked than horses, and donkeys are rarely affected.

In practice it is not customary to diagnose a case as one of strangles unless the patient has suppurative lymphadenitis of the submaxillary or the pharyngeal glands (abscess formation), but there is reason to believe this is not an essential lesion, and that only catarrh of the upper air passages may be present in some cases of the disease. In fact, in outbreaks of the disease in studs of horses, several cases of this form of the disease appear, and in many instances the disease has apparently been introduced into a stud by an animal that has been thought to have had nothing worse than an ordinary cold or sore throat.

The disease is usually one of young animals, but it may occur

in animals of any age, and frequently in South Africa, when an outbreak occurs, quite old animals will become affected. This raises the question of immunity, about which a difference of opinion exists. I have always thought that the reason for such old animals contracting the disease was due to them not having come in contact with it before, which could easily happen in South Africa, owing to the distances between places and the practical isolation of animals; but such a view is not shared by all, and may be ill-founded. In certain protozoal diseases, it is believed that the immunity only remains active and permanent if the animal is able to frequently become reinfected with the causal organism. Something similar may occur in strangles, and then it would follow that in large studs, and in towns where the disease is frequently present, aged horses would not visibly suffer from the disease a second time; but that animals removed from all contact of the disease for a few years might lose their immunity, and do so when again brought in contact with it.

The organism (*Streptococcus equi*) cannot be distinguished from the *Streptococcus pyogenes* except, perhaps, by Marmarek's test (growing it in broth in which other streptococci have previously been grown). It is destroyed by strong sunlight in a few hours, and by desiccation in a few weeks.

The common method of infection is probably by ingestion by the food or drinking water becoming contaminated by the discharges from the affected animal. It may also occur through inhalation or by means of a wound. In this respect the mouse is extremely susceptible; a mere scratch with a needle infected with the organism will usually kill it in three or four days. When a susceptible horse is inoculated subcutaneously with a culture of the organism, a localised abscess is provoked; when inoculated into the blood-stream a pyæmic condition is set up, and abscesses occur in different parts of the body, especially in the kidney, and frequently in the submaxillary and pharyngeal lymphatic glands, so that it appears as if these glands may be a predilection seat of the disease. There is no nasal catarrh, but acute general pleurisy or peritonitis may follow.

Infection by wounds is not of common occurrence in practice, but castration wounds have been known to become infected, probably by the hands of the operator. Foals may be born, at full time or prematurely, affected with strangles, and a foal may infect the udder of the mare, or the udder may infect the foal. Likewise a stallion may infect a mare during coitus, but

cases of this nature are rare, the great majority of infections occurring either by ingestion or inhalation.

Certain causes, such as colds, chills, fatigue, ill-ventilated and overcrowded stables, are regarded as predisposing causes of the disease. Perhaps in the past too much importance has been attached to these causes. In practice, probably the greatest predisposing cause is found to be bringing a number of susceptible horses together irrespective of their bodily condition or the hygiene of their surroundings.

In an outbreak of strangles in a large stud, in some instances the disease assumes two or more different forms. There are a certain number of cases in which the upper air passages only are affected; in other cases the typical abscess formation in the glands between the jaws and in the throat occurs, both of which forms are preceded and accompanied by a muco-purulent discharge from the nostrils. In exceptional cases, strangles become pyæmic in character, and abscesses may appear in either the kidney, spleen, or lungs, and in lymphatic glands other than the submaxillary and pharyngeal; and abdominal strangles may occur either with or without the distinct lesions in the jaw and throat glands. Abscesses are found in the mesenteric glands, and such cases often end fatally by the disease extending to the peritoneum. Another complication is a form of cerebral strangles. Abscesses which are probably an extension of the disease from the nasal chambers form in the brain.

Incubation.—The period of incubation of the disease is from three to five days as the result of experimental inoculation, but with natural infection it may be eight days or longer.

Symptoms.—Strangles frequently commences like an ordinary cold; there is slight fever and a discharge from the nostrils, the throat more or less swollen and painful, a difficulty in swallowing, particularly water, which runs out of the nose as the animal drinks. Concurrently with these symptoms there may be a swelling of the lymphatic glands (one or both) between the jaws, or this swelling may not appear for a few days, and a soft cough will be present. In mild cases no further symptoms than the foregoing may be exhibited. The swelling of the glands may disappear without abscess formation, and the animal returns to normal health in the course of a few days.

In other cases the symptoms become accentuated. The animal goes off its feed; the discharge from the nostrils becomes profuse and opaque; the glands under the jaw increase in size, are firm, hot, and painful when touched; a greater difficulty in

swallowing may be exhibited. In some cases the breathing is interfered with, and the animal makes a roaring sound. This is usually the case when abscess formation is going on in the glands of the throat. These abscesses are frequently deep-seated, and require several days before they are in suitable condition for opening, during which time the animal stands with its nose poked out, is unable to eat or swallow, and loses condition rapidly. In such cases there is the danger of complications arising by an extension of the disease causing inflammation of the lungs or pleura.

Comparison with Glanders.—In the great majority of cases the veterinarian has no difficulty in differentiating between a case of glanders or strangles, but cases happen where a difficulty in deciding occurs. Of course, there is the Mallein test, which will satisfactorily decide the matter, but this may not always be on the spot and available. In any case, it is well to know the salient differences, which are as follows:

Character of the Nasal Discharge.—In glanders this is sticky and adheres to the nostril, and is somewhat like starch in appearance. It usually comes from one nostril.

Character of the Swelling of the Jaw Gland.—In glanders this is hard and seems fixed to the jaw-bone. It is not painful or hot, the skin over it is loose, and not inflamed. It shows no tendency to disappear or to form an abscess. In strangles frequently, as well as the gland or glands, which are hot and painful and swollen, the throat is in a similar state, making swallowing difficult. The swelling of the gland is usually progressive, leading to abscess formation.

State of Health.—In glanders the appetite is good, frequently abnormally so, but an improvement in condition corresponding with the appetite does not occur. Urination is frequently profuse.

In strangles the appetite is usually impaired, but after the abscesses have burst the appetite returns and the condition improves. Urination is usually scanty.

Ulcers in Nostrils.—In cases of glanders presenting symptoms similar to those of strangles, ulceration of the nasal septum are usually found, and the discharge may be tinged with blood. In strangles, ulceration is also said to occur, but is rarely, if ever, seen.

Treatment.—Curative treatment depends upon the severity of the disease and the animal affected. With young, unbroken animals in many cases a policy of non-interference is the best

one to adopt; but in ordinary cases the animal should be isolated in a warm, airy box, if available, and put on light diet, such as bran mash and green food. Two to four ounces of Epsom salts may be given in a bucket of drinking water. It is inadvisable to attempt to drench animals suffering from strangles. In the first place, there is usually no necessity for doing so; and in the second place, owing to the soreness of the throat and difficulty in swallowing, there is a great danger of choking the animal, and through some of the medicine getting into the windpipe, of setting up complications which usually prove fatal.

For the throat a mustard blister or plaster will be found beneficial and give relief. Mix the mustard with water to about the same consistency that it is used for table purposes, and rub this in on both sides of the throat up to the roots of the ears and down between the jaws.

If the throat is very sore, an electuary may be given composed of extract of belladonna 1 dram, chlorate of potash ½ ounce, honey or treacle 4 ounces. Rub the belladonna down in glycerine in a pestle and mortar, then add the other ingredients to make a paste, and place about a teaspoonful with a flat stick on the tongue three or four times a day. If the breathing is bad through the swelling of the throat, steaming and inhalation (see article on Influenza) should be adopted. The thing that will give the greatest relief to the patient is the bursting of the abscesses, and endeavours should be made to " ripen " these as early as possible —that is, bring them to a head or point to get the pus to the surface as quickly as possible. To do this, one of several methods may be employed. One may use hot fomentations, and, between these, fastening a hot, warm sponge, tow, or cotton-wool, over the place by means of the headstall; or the abscess may be blistered either with ordinary fly blister, or the red biniodide of mercury blister, or the mustard may be repeated.

Usually, better results are obtained if the abscesses are allowed to burst naturally, but when they are situated in the throat (in the parotid gland) they are frequently deep-seated, the pus taking a considerable time before it can reach the surface. In such cases, to relieve the distress of the patient, it may be justifiable to open up the abscess with the knife directly the place it is pointing can be found. The pus will escape with considerable force usually from such an abscess. If this interference takes place, the wound must be kept open as long as possible by dressing it twice daily, and plugging it with tow to prevent it healing, the danger being that it will heal too soon, in which case it will

gather up again, and the same tiresome business will all have to be gone over again.

The abscesses should be washed with disinfectant and kept clean inside by syringing out. A solution of Jeyes, or other similar disinfectant, answers this purpose.

For some days many cases are unable to take any solid food, and liquids such as oatmeal or mealie meal gruel, linseed and hay tea, milk, etc., have to be relied on to maintain the animal's strength. But with the evacuation of the abscesses the appetite usually returns, and the animal recovers, but it should not be returned to work until it is thoroughly fit and strong, as there is a danger of inducing pneumonia if it is worked too soon.

In the pyæmic form of the disease, when abscesses form internally, very little can be done to assist the patient.

Prevention.—This consists in avoiding contact with infected animals or places that have been contaminated by the discharges from them. Care should be exercised in the introduction of a new or strange horse into the stable or stud, as it should in putting one's animals into a strange stable.

When the disease is present in a stud, endeavours should be made to prevent its spread by strict isolation of all infected animals, their attendants, and utensils used for them; disinfection of mangers, water-troughs, buckets, and everything that may have become contaminated by the discharges.

A valuable serum can be obtained from horses hyper-immunised against strangles. This has been used both as a curative and a preventive. The only drawback to its general use is that it is rather expensive, and that it only gives protection for one or two weeks; therefore it has to be repeated to tide animals through an outbreak of the disease. But in valuable breeding studs such expense is justified to protect the young stock during an outbreak. Perhaps I should mention that one of the dreaded sequels of strangles to the owner of thoroughbred horses is that the animal may be left a "roarer" or "whistler." These diseases are rarely met with in South Africa. The dose of serum is 20 to 30 c.c. once a day as a curative, a similar dose at intervals of one or two weeks being used as a preventive.

CATARRH

Synonyms.—Common Cold, Contagious Cold.

Nature.—Catarrh is indicated by sneezing, running from the eyes, redness and dryness of the mucous membrane of the nostrils, succeeded by a discharge, at first watery, which soon, however, becomes turbid, yellowish-white, and profuse. It is associated with a varying degree of fever, dulness, and debility.

As previously mentioned, practically all cases of strangles with abscess-formation have nasal or bronchial catarrh as an accompanying condition, but in a great many cases of febrile catarrh in young horses brought to large cities there is no abscess formation developed, although the cases may be numerous and although the malady has every appearance of being contagious. The cause of this catarrh has not been determined. The nasal and bronchial discharge contains streptococci in large numbers, and frequently pure in culture. The animal often develops a pneumonia or a pleurisy, and these lesions also contain the streptococci, apparently contagious, but distinct from strangles. This disease is more frequently seen in towns and large studs of animals. It is rarely seen in the country districts.

PNEUMONIA, OR INFLAMMATION OF THE LUNGS

Synonyms.—Contagious Pneumonia or Pleuro-pneumonia of the Horse, Stable or Hospital Pneumonia, Pulmonary Influenza.

Nature.—" In most textbooks, contagious pneumonia of the horse is placed amongst the infectious diseases, while ordinary or sporadic pneumonia occurs under the heading of Respiratory Affections. This would seem at first sight to indicate that the two affections were distinct from each other, both pathologically and clinically. We must point out, however, that all pneumonias depend on microbic infection, and that the causes formerly accepted—viz., exposure to cold, chills, fatigue, etc.—must be regarded as predisposing to the disease, and not as direct etiological factors " (E. Wallis Hoare). But as it is not known what bacterium is actually the cause of pneumonia, opinions offered upon the matter can only be regarded as speculations. In this connection, it is contended that the differences in clinical manifestations of pneumonia depend upon the virulence of the micro-organisms present. It might also be held that the degree of

susceptibility or the natural resistance of the individual would influence the symptoms presented.

The disease that is usually recognised as contagious pneumonia in the horse is apparently a distinct affection, the chief character of the lesions being a tendency to necrosis (death) of the hepatised lung tissue. This generally sets in at a number of separate centres, and is usually followed by putrefaction of the necrosed areas. It goes from the dirty-white, solid character of necrosis into a greyish material sometimes like pus, and is often erroneously described as abscess of the lung. In a large proportion of cases this putrid pneumonia is followed by a putrid pleurisy. The cause is difficult to trace on account of the rapidity with which necrosis and putridity occur. The diseased lung is quite a menagerie of bacteria, and the prime offender cannot be determined; it may not be present, as it may have been crowded out by that time by others. As a result of investigations, several organisms have been suspected. The two that have, perhaps, the greatest number of adherents are a pasteurella and a streptococcus, but not the strangles streptococcus. It may be the same streptococcus that causes nasal catarrh.

Some authors regard this gangrenous pneumonia as a purely and highly contagious disease, but this is not certain. It apparently may be, and is sporadic, but there is no doubt, however, as to its contagiousness in certain characters.

Symptoms.—Temperature 104° to 107° F. The pulse is usually quickened. If it is strong, it is favourable to recovery, but a weak, soft, or irregular pulse is the reverse. The mucous membranes are congested, the breathing accelerated. The difficulty of breathing will usually denote the amount of lung tissue involved, but not always so, as cases occur in which respiration is but slightly affected. A cough may be present, but, as a rule, the animal is afraid to cough on account of the pain produced, and the cough is suppressed to as great an extent as possible. The animal is usually much depressed, with a hanging head, loss of appetite, and presents an anxious appearance. In other cases the animal may appear to be fairly cheerful, retaining a certain amount of appetite, and apparently progressing favourably, when pleurisy, which is a frequent complication, sets in. The animal then fixes its ribs firmly, and uses the abdominal muscles in breathing to as great an extent as possible, causing a ridge, extending from the hip along the ends of the ribs to the breast bone, to become fixed and prominent. The animal will evince a great soreness if pressure is applied by the fingers

between the ribs. This is quickly followed by an exudate from the pleuræ into the cavity of the chest. With the pouring out of the exudate a temporary relief from the pleural pain occurs, but the difficulty in breathing will be increased in proportion to the quantity of the exudate in the chest cavity. The amount of exudate present may be determined by percussion—that is, by placing the left hand flat on the ribs and tapping the fingers of the left hand with the fingers of the right, moving the hand from below upwards until a difference in the sound denotes that the top of the water-line has been reached. In fact, the process may be likened to tapping a corrugated iron tank with the fingers to find out what depth of water there is in it. The water-line will be found to be at the same level on both sides of the chest. To confirm the diagnosis of the extent of the fluid, if the ear is applied to the chest wall, an absence of respiratory sounds will be found to exist up to the level of the fluid.

Changes in the state of the patient occur rapidly in many cases in this disease. This pouring out of the pleural exudate may come on suddenly and proceed quickly, and after it has occurred, or in those cases in which it does not occur, the patient may appear to be not in a really serious condition, yet on the next visit to examine it, on entering the stable one is assailed with a horrible smell, the odour of which there is no mistaking, denoting that gangrene of the lungs has set in. Animals that have apparently recovered from the disease, and that have been put to work too soon, have been known to develop gangrene of the lungs and die within a short space of time (one or two days).

When gangrene sets in, a marked change occurs in the animal, as a rule. The temperature rises; the animal is greatly depressed, and wears a very anxious expression; it loses its appetite entirely; the pulse becomes small, weak, and "running out" almost or quite imperceptible; the respiration troubled with a greyish fœtid discharge from the nostrils. These symptoms will vary in intensity according to the probable termination of the cases. Animals do recover, and others put up a big fight against the disease; in such the symptoms will not be so marked.

As in cases with pleuro-pneumonia in cattle, when horses suffer from this disease, or, in fact, any disease of the chest cavity, they usually remain on their feet all the time, or as long as they can, and rarely or never lie down.

Treatment.—In considering the preventive treatment of the disease, one will be greatly guided by the history of the stable in which it occurs. The disease is one of great cities and large

studs. It does not occur to a great extent in South Africa; therefore, in the latter country, the precautions that should be taken in the former places will not be applied. In stables where the disease has occurred, it is advisable to isolate all new purchases for a few weeks; to isolate immediately any animals showing symptoms such as a feverish cold or cough; to thoroughly disinfect the stables, particularly the stall or box in which cases have occurred, and generally to put the place in as good a hygienic condition as possible.

With regard to curative treatment, in my experience the greatest help towards successful treatment of the disease is fresh pure air. Cases in dark, ill-ventilated stables in towns are nearly always fatal; but if similar cases can be removed to the country, and obtain fresh air and good hygienic surroundings, many will recover. Acting on this knowledge, I have employed oxygen gas as an inhalation with marked if but temporary benefit.

Next in importance to fresh air comes good nursing, which does not only include tempting the appetite with food, but also attending to the bodily comfort of the patient: keeping the extremities warm by bandaging, rugging the animal up, changing these occasionally, sponging the nostrils, and in other ways trying to freshen the patient up.

Drenching of the patient with medicine is attended with danger, and little doubt exists but that many cases of septic pneumonia are the result of some of the medicine getting into the windpipe when drenching animals. In the early stages of the disease the patient should be treated in a very similar way to that recommended for strangles and influenza. Epsom salts may be given in the drinking water, and if it is desired to give febrifugal medicines, this may be done in the form of an electuary such as nitrate or chlorate of potash with some liquorice powder mixed up with honey or treacle and placed on the tongue. Inhalations of turpentine, eucalyptus oil, and carbolic acid, as applied in the before-mentioned diseases, will at times afford relief, but a cardinal principle is to do nothing to distress the patient.

Later on in the disease, after six or eight days' illness, and where the foregoing treatment does not appear to be having the desired result, and the case is hanging fire, and gangrene of the lung may be anticipated, it has been recommended to inject 2 drams of formalin in 8 ounces of water into the jugular vein. To inject such an amount of fluid, the injection has to be done very slowly. It can be done with an ordinary large-size hypodermic syringe. Get the vein up with a cord around the neck in the

ordinary way, insert the needle in the vein as soon as this is done, and the blood spurts in a full stream through the needle. Release the cord and slowly inject the mixture.

Nuclein solution in doses of 25 to 30 c.c. injected into the vein once daily for a few days until the temperature is brought down to about 103° F. has also been used with success. The solution should be warmed prior to injection.

Normal salt solution injected into the vein is also highly spoken of as a heart stimulant in cases of pneumonia.

I have had good results from the employment of tallianine in lung cases in calves, and think this drug is well worthy of a trial in pneumonia in the horse.

With regard to the benefit derived from the application of counter-irritation in pneumonia a difference of opinion exists. I have thought it has done good in most cases; in others I have been sceptical about the benefit following its application, but I usually employ it in the form of ½ pound of mustard mixed with water and rubbed on the ribs on both sides of the chest, and covered with a sheet of brown paper and left there.

The treatment of the case when an effusion has taken place into the chest has to be directed mainly to keeping up the animal's strength in the hope that the fluid will become reabsorbed. In such cases the employment of nuclein is indicated. I have tapped the chest and drawn off this fluid in a few cases, but as I found no benefit to follow from doing so, discontinued the practice and painted the chest with tincture of iodine instead, from which I think I have obtained better results.

PLEURISY, OR INFLAMMATION OF THE PLEURA

Pleurisy is usually a secondary affection, but cases are met with in which the disease develops in the absence of any appreciable cause. For the purpose of description, according to its course, the disease is frequently divided up into a variety of forms, such as acute sero-fibrinous pleurisy, purulent pleurisy or empyema, fibrinous plastic or dry pleurisy, chronic pleurisy, etc.

From a bacterial point of view, perhaps, it is better to divide cases of acute and subacute pleurisy with abundant effusion into the chest cavity into (1) putrid pleurisy, (2) non-putrid pleurisy.

Apart from pleurisy due to wounds, all putrid pleurisies are secondary to putrid pneumonia, and the liquid exudate and the pleura swarm with bacteria of different kinds. Cases of this form

of pleurisy are frequently met with in the disease just described—contagious pneumonia of the horse—and also in lung-sickness in cattle.

Non-putrid pleurisy is usually a streptococcic pleurisy, and the lymph contains great numbers of streptococci in pure culture; therefore, the streptococcus is regarded as the actual cause of the disease. The liquid exudate is often poor in streptococci, and is confined to the solid fibrinous exudate.

Pleurisy is frequently associated with a number of diseases, such as pneumonia, influenza, strangles, glanders, tuberculosis, rheumatism, inflammation of the heart and heart sac, etc. Dry pleurisy is often seen in tuberculosis. Adhesions of the lungs to the ribs is frequently met with in the same disease, also in lung-sickness in cattle. Pleurisy may also be unilateral or bilateral, as it affects either one or both lungs.

The pleural exudate varies in amount and in general character. In perhaps the majority of cases it is yellowish in colour. It may be turbid, blood-stained, brownish, dark-coloured; it may be purulent and fœtid. It frequently has masses of fibrin floating in it, somewhat resembling the white of an egg when poached. Similar masses are attached to the lungs and ribs.

It must be remembered that it is an inflammatory exudate, and distinct from that which occurs in hydrothorax (dropsy of the chest), which is a non-inflammatory exudate and does not contain any fibrin.

ACUTE RHEUMATIC AFFECTIONS

Rheumatism is a term that is rather loosely applied to obscure forms of lameness. The acute forms of the disease affecting the joints are regarded by many authorities as an acute infectious disease depending on an unknown infective agent.

Rheumatic fever in the human agent is an acute but non-suppurative inflammation of the joints and endocardium (the lining membrane of the heart). Exactly similar cases occur in the lower animals; similar cases occur in swine erysipelas. Cases are rare in the horse. I only remember having seen one such case in a colt that was recovering from strangles. In the horse the joint and heart lesions contain streptococci. The same is true for man, but considerable differences of opinion exist as to whether these are the actual cause of the disease; therefore, I do not purpose to describe the disease fully under this section.

MAMMITIS, OR INFLAMMATION OF THE UDDER, IN THE COW

Synonyms.—Mastitis, Garget, Weed, Mastitis, Catarrhalis Contagiosa.

This very common disease in the cow appears to be caused by streptococci, but a number of organisms have been found frequently in the same case. In fact, it is the rule to find mixed infections; therefore, it is possible that more than one organism is responsible. The disease exhibits very different clinical characters in different cases. These differences may perhaps be accounted for by different organisms, or organisms of different degrees of virulence, being present.

For one to assert that all cases of mammitis are contagious might be considered rather rash, in view of the fact that single isolated cases of the disease frequently occur in a cow-shed. Nevertheless, present-day knowledge of the disease points to the probability of the assertion being true, and that the reason why the disease in such cases has little or no tendency to spread can be accounted for by the organism (streptococcus ?) producing it being a facultative parasite of mild virulence. On the other hand, there are cases of markedly contagious mammitis in cows caused by streptococci. In these cases, the inflammation is frequently not so intense or acute, and in some cases the alterations in the gland tissue are comparatively trivial. There is little or no swelling, sensitiveness, or heat present.

The classification of mammitis caused by pus-forming organisms is somewhat difficult. Some authors arrange their classification according to the virulence of the attack as (1) subacute; (2) acute; (3) chronic; but as I purpose to include cases of mammitis caused by other organisms, my classification will be somewhat different.

Nature of the Disease.—For over a century mammitis has been recognised and written about as a serious disease of stock, and for a long time it was believed by some veterinary practitioners to be contagious; but it was not until 1877 that Zarn actually

contended the disease was contagious, and not due to the causes usually assigned.

Cases of the disease have been known to occur in dry cows, but usually it occurs in cows when in full milk, heavy milkers being more susceptible, and cows of this description with leaking teats perhaps more so than any others. This may be accounted for by the belief that the commonest route of infection is by way of the teat canal. At first sight this may appear a somewhat improbable way of entrance, but when it is remembered that before the organism causing the disease can be seen it has to be examined under the oil immersion lens of the microscope, which magnifies over a thousand times, it does not do so, but suggests some such an idea as a mouse going into a railway tunnel. Hard milkers or animals in which the sphincter muscle closing the orifice of the teat is strong are not so liable to infection by this way, but the cow with a weak sphincter that allows the milk to flow freely, or that allows a drop to escape and hang on the end of the teat before she is milked out, offers a favourable medium in the milk for growth, and an easy ingress to the organism. Other routes of infection are by the skin, which, being very thin on the udder, is liable to become broken. Such broken places may constitute starting-points of infection. It is possible that the disease in natural cases may occur by the entrance of the organism through the unbroken skin. Kitt painted the teat with a pure culture of the organism, and mammitis resulted. Another route of infection is by the blood-current or lymph-stream. Such route is taken in tubercular mammitis. It is rare for pyogenic bacteria to reach the udder in this way.

Seeing that the disease is in the great majority of cases caused by pus-forming organisms, not only every case of the disease, but also every suppurating sore in the cow-shed, should be regarded as a possible source of spreading the disease.

As a general rule there is relationship between the acuteness of the attack and the contagiousness of the disease; the more severe the type, the greater the infectivity. In this connection it is well to remember that the disease is, in the majority of cases, spread by the hands of the milker; therefore, under ordinary circumstances, its spread is, as it should be, slow when compared with many other contagious diseases. If a fresh case occurs every day for some days, it is regarded as a virulent type of the disease. Frequently several days intervene between the cases, or there may be only one case in the herd.

Simple Mammitis.

Cases of congestion of the udder occur particularly in deep milkers just before or shortly after calving. Many of these cases, if taken in time and treated properly, may be said to never pass beyond the stage of congestion; but, on the other hand, if neglected or wrongly treated, a very severe inflammation may result.

Particular care should be given to cows with big bags to see that the udder is not injured, and is guarded against draughts and chills caused by lying on cold, damp ground or concrete, as these must be regarded as predisposing causes of the disease, and we know that the pus-forming organisms lurk plentifully in many places. If the udder is particularly heavy and pendulous before calving, it is sometimes advisable to support it by a sheet. Pass this under the udder and tie it over the back after having cut holes in it to allow the teats to come through. If this condition prevails after calving, the cow should be frequently milked; and if the udder is hard, it should be hand-rubbed. Another plan is to put a hefty calf on the cow, if necessary. Some of the milk may be drawn before doing so, so that there shall not be a too plentiful supply for it, which will cause it to " bunt " the udder with its head.

Another precaution to be taken with such cows is to put them on a laxative or spare diet, and withhold all rich and heating food. Plenty of coarse salt should be supplied them; some owners also give one fairly large or two or three small doses of Epsom salts.

Symptoms.—The animal is not seriously indisposed. She will probably be off her feed a bit, but will still eat something and chew the cud; the temperature will be raised 103° to 104° F. The symptoms do not appear very suddenly, and the milk is not greatly reduced in quantity; and in the early stages of the disease it may be but little altered, if at all, in appearance when spread out in the palm of the hand in as thin a layer as possible for examination. If no alteration in the milk has taken place, and suitable treatment is immediately adopted, the chances are in favour of a speedy recovery; but if small lumps of casein about the size of pin-heads or larger are seen, recovery will take longer —probably about a week.

With regard to the udder itself, usually only one quarter may be affected, but two may be. In the early stages little or no swelling and pain may be present, or it may be, with a reddening of the

skin, which may disappear in a day or two; or it may increase within the same time, and be attended with a considerable change in the nature of the milk, which becomes yellow and watery, and contains clots difficult to milk out.

After recovery a slight thickening of the affected quarter may remain, and less than the normal amount of milk may be given from that quarter until after the next calving.

Traumatic Mammitis.

Under traumatic mammitis is included all forms of the disease due to injuries, which may consist of a simple bruising or broken skin. The ways that injury can be caused to the udder are many; amongst the commonest may be mentioned horning, and by a cow stepping upon her own teat, or being stepped upon by another cow. In such cases two sets of conditions have got to be treated. In the first place, every endeavour should be made to allay the inflammation and prevent its extension. For this purpose the cow should be given a dose of Epsom salts, and put on a cooling diet, and the injured part should be well bathed with warm water. To get the correct temperature of the water, one should be able to hold one's elbow (not hand) comfortably in it, to which a small quantity of disinfectant has been added, such as carbolic acid. After the bathing, the udder should be thoroughly dried with a soft towel, then something to keep the skin soft and supple should be applied, and that will prevent it drying and becoming painful to the touch. Camphorated oil is good for this purpose, but if it is not at hand, lanoline, zinc ointment, or carbolised vaseline may be used.

If the injury is near the end of the teat, there is a danger of the closing of the milk duct by adhesion of its injured walls. To prevent this a flexible lead probe of sufficient calibre to stretch the opening a bit should be placed in the canal, and kept there all the time, excepting when the teat is being milked, until the danger of adhesion is past. The probe is kept in position by running a tape through its eye and then being wrapped loosely round the teat. A teat syphon will answer the purpose, but the hole in it should be blocked up to prevent the exit of milk as well as the ingress of organisms. Needless to say, whichever is employed, it should be sterilised by boiling before use each time.

If the injury is upon the udder itself, or should the extension of the inflammation cause the milk to become watery, bloody, or thick, the only thing to do is not to lose patience, but continue

the bathing and milking, in the hope of saving the quarter for future lactations. At times this is impossible, as abscesses may form in the gland, or a wound may have penetrated quite into the gland, when gangrene may set in, and the whole gland of the quarter sloughs away and has to be removed. In the case of abscesses, the gland is usually destroyed and is henceforth functionless. The affected quarter withers up after the animal has recovered.

Acute Mammitis.

The onset of this form of the disease is sudden. A cow may appear quite normal when milked in the evening, but in the morning is found standing, perhaps shivering, with the coat staring, the temperature elevated to 105° F., and the animal evincing great pain in the udder by straddling and frequent movements of the hind legs to relieve the udder from lateral pressure.

The appetite and digestion is considerably upset, the cow is entirely off her food, and ceases to chew her cud; the bowels may be constipated, and the stomach slightly distended with gas. In other cases diarrhœa will be present.

The affected portion of the udder—usually one, but in some cases two quarters—is swollen and hard, reddened and shiny, very hot and very painful when touched. Little or nothing in the shape of milk can be extracted through the teat. A few ounces of yellowish-brown coloured, watery fluid may be obtained later on. This fluid becomes thicker and more stinking. As the case progresses the fluid becomes more and more pus-like.

After two or three days the systematic disturbance abates somewhat, the animal commences to feed again, and the pain in the udder decreases. The product obtained from the quarter may have increased in quantity, whilst maintaining its pus-like appearance, and the animal may continue to improve slowly until quite recovered, but usually with the loss of function of the affected quarter for all time. On the other hand, the teat may become so hard that it is impossible to draw off the fluid, and a change in colour of the teat takes place. It first becomes violet-red, which deepens in colour to a greenish-black. The teat becomes cold, and when drawn gives off a dark-red, evil-smelling fluid. This indicates that gangrene or death of the part has set in. That will quickly affect the entire gland in that quarter. The animal may show very distressing symptoms, and die within a few hours from septic intoxication; or the skin may slough and

the gland come away or be removed intact as a dead fœtid mass of tissue.

In other cases the gland remains hard, more or less, from the start of the disease until an abscess or abscesses have ripened and burst, or been lanced or relieved by cutting off that teat at its juncture with the udder. In the majority of cases of acute mammitis the result is the loss of the quarter affected, and it is in this form of the disease that its contagious nature by further cows in the herd becoming affected is frequently observed.

Infectious Mammitis.

By this term I wish to include a form of the disease that I have never observed in South Africa, but that I saw in the West of England. The disease in these cases was of a markedly contagious nature; the inflammation was not so intense or acute, and in some cases the alteration in the gland tissue was comparatively trivial. There was little or no swelling, sensitiveness, or heat present; but the milk is more or less altered before it leaves the cells, and from early in the attack is pus-like and offensive.

Treatment.—The preventive treatment of mammitis consists in cleanliness and disinfection. Cases of the disease are more frequent in herds that are housed in dirty sheds, or where the system of treatment and milking of the cows is carried out in a slipshod, dirty manner.

Cow-sheds should at least be thoroughly cleaned and disinfected once a year; the udder should also be swabbed clean before milking; but perhaps the matter of greatest importance is the condition of the milkers' hands. A person who has been dressing wounds should not be allowed to milk cows, but, if he must do so, the hands should first be well washed in disinfectant. No animal with suppurating sores of any description should be allowed in the cow-house, but should be isolated. So should all cows that are suffering from the disease. Likewise cows with retention of the afterbirth should not be stabled amongst the cows in milk, and particular attention should be paid to prevent the discharge from the fœtal membranes from reaching the udder of the cow so affected. Cases of mammitis frequently follow in the animal that has had retention of the afterbirth.

The affected quarter of the cow suffering from the disease should not be milked on to the floor, as is so commonly done in this disease, but into some vessel from which the product can be destroyed. A good plan is to milk it into a bucket containing

a strong disinfectant. If possible, it is advisable to have a separate milker for affected cows, but if he must milk healthy cows as well, he should milk them first and the diseased animals last.

Medicinal treatment is of great service in the milder forms of the disease, but does not help to such a great extent in the severe forms. A bold dose of aperient medicine, such as a pound of Epsom salts with a dram of ginger added to it, seems to lessen the force of the attack. Some practitioners favour the use of aloes as a purgative in this disease, and will add 1 ounce or even 2 ounces to the salts and the ginger for a big robust cow; but, whilst a good purging out is beneficial, it must be remembered that it can be overdone and harm result.

The next thing is that the animal should be made as comfortable in her quarters (stall or box) as possible. If she is in the shivering stage, she should be rugged up, and a cupful of dop brandy or other stimulant may be given, mixed with sufficient water to dilute it. After the preliminary purgative, some practitioners discard the further use of medicine by the mouth; others like to give 2 ounces of hyposulphite of soda in water daily. Camphor is a drug that has been long in use, because it was believed to be excreted by the udder, and it is recommended to be given in 5-gramme doses. Formaldehyde has been found most beneficial both in Europe and America. It is given in a daily dose of $1\frac{1}{4}$ drams of 40 per cent. formaldehyde well diluted with water.

The treatment of the udder itself at the commencement of the disease should consist of hot fomentations, gentle and prolonged massage of the udder with the hands, followed by stripping of the teat to remove the retained secretion. The foregoing are the broad lines upon which to work. Of course, they have to be varied to suit individual cases. If nothing can be got from the teat, it is no use in increasing its soreness by repeated attempts at stripping. Likewise, if the udder is as hard as a board, it is no use making it more painful by rubbing it with the dry hand, but some greasy, penetrating preparation is required. A number are used. Amongst the best that I have seen are the old-fashioned ones of goose fat or lanoline, which is the grease of sheep's wool; but these cannot always be obtained, or, even if they can, I think a better agent to employ is camphorated oil, and this can be further improved by the addition of belladonna to it. Say, rub $\frac{1}{2}$ ounce of extract of belladonna down in 1 to 2 ounces of glycerine, and add this to 4 ounces or more of camphorated

oil. The udder should always be rubbed with the camphorated oil before leaving it.

The position of the udder does not lend to poulticing, but if it is necessary to put on a suspensory bandage in the form of a sheet to support it, as already described, cotton-wool should be packed in around the udder.

Irrigation of the udder is successfully employed by some practitioners; with others it has not found favour. The agents recommended are a mild solution of peroxide of hydrogen, or a solution of corrosive sublimate 1 to 3,000 of water, or a 4 per cent. boric acid solution at body temperature, leaving it in the udder for about four hours before drawing the teat.

In addition to the foregoing treatments, painting of the affected portion of the udder, teat included, once a day with tincture of iodine has been found to do good; or, in place of the tincture, ordinary iodine ointment can be rubbed in.

In certain cases, in spite of the foregoing treatments, the udder still remains hard and indurated; but the acute pain and redness of the skin has disappeared. In such, a mild application of ordinary red blister (biniodide of mercury) should be rubbed in.

In cases where it is impossible to remove the pus or clotted material by the teat, and where the practitioner feels confident that even if the case ends in the most favourable way it will mean the total loss of that quarter, no time should be lost in removing the teat by cutting it off close up to the udder. Such a procedure simplifies treatment considerably. In the first place, it gives perfect drainage, which may be assisted by massage coupled with frequent irrigations of weak antiseptic solutions.

Tubercular Mammitis.

This form of mammitis in the cow in South Africa is perhaps rare when compared with the frequency with which it is found in some other countries. This comparative rarity is probably due to cases of tuberculosis being in fewer numbers, and not to any difference in the nature of the disease.

It is a matter of serious importance from a public health point of view, as a tuberculous udder is one of the surest methods of spreading contagion to man or any animal that drinks the milk from such an udder. Of course, no treatment may or should be resorted to. All such infected animals should be destroyed.

The frequency of tubercular mammitis in the cow is one of the outstanding features of bovine tuberculosis. In the human

subject it is exceedingly rare, and it is rare or unknown in all species of the domesticated animals other than the cow. Cases have been met with in the sow. The frequency of its occurrence in the cow is probably due to the frequency of peritoneal tuberculosis in that animal.

Much discussion has occurred as to the way the infection reaches the udder. The two views that are in dispute to the greatest extent are as to whether it occurs by the blood-stream or by the lymphatics. The facts appear to be greatly opposed to the blood-stream view being the correct one, as at the onset of the disease it mostly involves only one quarter of the udder, and in that quarter it is not in the form of a miliary tuberculosis; and in the majority of cases there is no evidence of a miliary tuberculosis in other organs of the body, such as one would expect to find by blood conveyance of the organism. The quarter affected is mostly a posterior one, and the disease usually commences at the top of it. It is not rare to find one or more supramammary lymphatic glands diseased whilst the udder tissue is still healthy, and in nearly all cases extensive peritoneal tuberculosis is present. These facts appear to be overwhelmingly in favour of the view that the disease reaches the udder by means of backward (centrifugal) lymphatic infection, and that the tubercle bacilli are carried by the lymphatics from the infected abdomen to the supramammary lymphatic glands, and from thence to the udder tissue.

Nature and Symptoms.—Tubercular disease of the udder is subacute or chronic in character. As a rule, it commences in an insidious manner and progresses slowly. Occasionally the disease assumes more acute characters; then the enlargement is more rapid and more marked. It usually commences at the upper part of a posterior quarter of the udder. It extends and invades the greater part of that quarter, and may spread to one or more of the other quarters; or, as I have already said, the supramammary lymphatic glands may be first affected and be slightly enlarged, and gradually increase until they reach the size of a child's head. Other favourite sites are just behind or in front of the teat, the tumour causing the quarter to sag. In other cases it may be observed that, after the udder has been milked dry, one can feel several nodules. These little tumours are firm and painless, and may remain apparently harmless for a long time.

The lesion is a painless one; it is not sensitive to pressure; there is no increased warmth of the part or vascularity of the skin; it steadily adds to the density of the affected quarter; and when the lesion becomes extensive, the quarter feels fibroid. Its borders

are well defined, though often irregular, giving it a nodular feeling. In very old cases it becomes almost stony in hardness. If the disease is uncomplicated, it never has a tendency to burst outwards.

Milk.—Even for some time after the lesion has attained an appreciable size the milk may not be sensibly altered in amount or quality. Sooner or later, however, the milk becomes poorer, and when centrifuged there is less cream, and an appreciable deposit in which, by means of the microscope, the tubercle bacillus can be demonstrated. As the disease advances the milk becomes thinner and gives more deposit, and if the animal survives, the milk becomes profoundly altered in appearance. The liquid is like whey, and contains strings of suspended material.

On post-mortem examination the most striking naked-eye character of an area of diseased tissue is its remarkable stiffness. It is not flabby at all like the healthy parts of the udder. There is an exaggerated appearance of lobulation, which is very indistinct in the healthy udder. It looks like coarse lobules half the size of a finger-nail.

The texture of the lesion is opaque. One rarely finds softened areas, or caseation, or calcification present, but this may be met with in old-standing cases.

Actinomycotic Mammitis.

Numerous investigators have shown that the udder of the cow and the sow is occasionally attacked by actinomycosis, and the route of infection is apparently by way of the teat canal, constituting what is frequently apparently a primary infection.

The disease is manifested usually by a hard swelling that cannot easily be distinguished from a tubercular infection in some part of the udder. The swelling increases in size and hardness, irrespective of what local treatment may be applied. In course of time pus centres may form which break down into abscess formation. When it has reached this point there is danger of other quarters of the udder being involved.

If microscopical diagnosis is made early, the iodide of potassium treatment (see Actinomycosis) will arrest the progress of the disease, and if the value of the animal warrants the operation, excision of the growth may be attempted.

GANGRENOUS MAMMITIS IN EWES

Synonym.—Black Udder.

History.—The disease has been recorded for over fifty years on the continent of Europe. It occurs also in Great Britain, but more rarely, the last outbreak there being in 1918. It has been observed in New Zealand.

Nature.—It is caused by a very small micrococcus discovered by Rivolta in 1875, and since thoroughly studied by Nocard.

The organism gains entrance to the udder either by way of the teat canal or through abrasions of the skin, the latter becoming infiltrated, and a doughy swelling of the udder follows; then a gaseous condition and gangrene, or black death, of the part, quickly followed by acute septicæmia and death.

Symptoms.—The symptoms are very acute. Usually one quarter of the udder only is affected. It is hot and painful to the touch, and appears engorged. The milk is first yellow and then red; next, patches of skin appear to be coloured dark violet, and these extend. From the painful condition, the udder quickly passes to insensibility, is somewhat shrunken, and feels cold—lifeless, in fact. The ewe is doomed. The signs of blood-poisoning (acute septicæmia) are evinced. She goes off her legs, shivers, grinds her teeth audibly. The temperature of the body falls below normal, and she dies in two or three days.

In the few cases that recover spontaneously the affected part is sloughed off, but does not heal in the manner of such cases when due to ordinary mammitis. Unhealthy ulcers remain and heal most reluctantly.

Treatment.—This must be of a preventive nature by immediately isolating all suspects, moving the herd off the tainted ground, disinfecting the kraal, or erecting a new one on a clean site. All articles employed should be disinfected, and attention should be bestowed upon the attendants as well. So rapid and fatal is the onslaught of the disease that no treatment offers any hope of saving the individual except excision of the affected quarter. This can be done by simply dissecting away the diseased part and ligaturing the bloodvessels. Others prefer burning the quarter off with a very hot iron. The latter method would appear likely to give better results; it sounds rather brutal, but if the part is dead and senseless it would not be so. Bridré was able to immunise sheep by growing the organism in bouillon and using it as a vaccine. He also found that hyperimmunised sheep possessed a serum having preventive properties.

MAMMITIS IN MARES

The disease is of much less frequency in mares than it is in cows. Occasionally cases of simple mammitis are met with in the mare that yield to similar treatment to that adopted for the disease in the cow. Of course, this difference exists that arrangements have to be made for hand-rearing the foal, a more difficult matter than in the case of a calf.

Perhaps the majority of cases of mammitis in mares end in abscess formation, or gangrene and sloughing of the part. To a certain extent this may be due to the disease not being discovered early, or the difficulty of treating such animals, but probably is due in many instances to the disease being contracted from some diseased condition of the foal, such as strangles or other suppurating sores.

MALIGNANT ŒDEMA

Synonyms.—Traumatic Spreading Gangrene, Hospital Gangrene, Gangrene (French, *Gaseuse*).

Nature.—The disease is caused by the malignant œdema bacillus, an organism that structurally greatly resembles the anthrax bacillus, and at the present day this resemblance is perhaps the matter of most importance in connection with the disease to the veterinarian and bacteriologist, as the determination of whether anthrax was the cause of death of an animal is frequently rendered difficult by the presence of the malignant œdema bacillus in the carcase.

In preaseptic days the disease was known as hospital gangrene in human practice, and was relatively frequent both in human and veterinary surgery; but now, owing to the general practice of aseptic or antiseptic methods, it has become an uncommon disease.

The organism is a facultative parasite, and its common method of existence is as a saprophyte in soil. It is especially abundant in cultivated and manured soils. The spores are thus necessarily taken in by herbivora with their food, and therefore the bacillus is a perfectly normal inhabitant of the alimentary canal of these animals. It is doubtful if it multiplies to any great extent in the alimentary canal, but the spores are present in the fæces.

The organism is principally confined to the lesions. Very few are found in the blood of the living animal, but immediately the animal is dead it starts to invade the carcase. This invasion starts from the alimentary contents, and is especially rapid in asphyxiated animals, and not so rapid in animals shot or destroyed whilst healthy. The invasion follows the lines of the veins, and the abdominal and thoracic organs are soon pretty completely invaded in any weather. The propagation of the organism is, no doubt, favoured by the carcase temperature. When the invasion reaches towards the peripheral parts of the body it is slower, because by that time the temperature of these parts has fallen considerably. The interval that elapses between the death of the animal and the organism reaching the extremities

of the carcase largely depends upon the external temperature. In temperate climates it occurs within twenty-four hours; in a tropical climate in less time.

The organism is a good example of the strictly anaerobic bacteria. It is exclusively as a wound infection that one meets with cases of malignant œdema. All the domestic animals, except the ox, are susceptible to the disease, but no animal is susceptible by ingestion or inhalation. Experimentally, the most certain method of infection is by intramuscular injection. The disease is chiefly met with in the horse as a complication of surgical or accidental wounds.

Symptoms.—The lesions consist of a rapid, ill-defined swelling around the wound that is sometimes emphysematous, and on post-mortem examination of such a case it is found that the swelling is due to an abundant, watery, blood-tinged liquid. Frequently the diseased part has a more or less putrid odour. In reality, cases of pure malignant œdema with the malignant œdema bacillus pure in the lesions never occur. The microbial invasion of the wound is a mixed one, staphylococci, streptococci, and putrefactive bacteria as well being present. The swelling, which is at first doughy to the touch and very hot, spreads with great rapidity, and whilst the outside portions of the swelling retain these characters, the central portions become softened, colder, and less painful—in fact, are quickly becoming "dead." The discharge from the wound is usually at first yellow, or reddish and watery; later on, it takes on a deeper colour, becomes frothy and putrid.

The animal goes off its food, the temperature rises to 104° F., the mucous membranes are injected, the pulse and breathing are accelerated. Very soon the animal appears to be in a very bad way. It has a very anxious expression, and is very dejected, with the head hanging down or resting on the manger, and sweats over different parts of the body, rapidly loses flesh, becomes very unsteady on the legs. The hairs of the mane are easily pulled out. The latter used to be a favourite test amongst stable-men as to the nearness of the end, which occurs in four or five days.

Treatment.—The preventive treatment lies in irrigating accidental wounds out well with some antiseptic fluid, and by the application of antiseptic dressings, such as iodoform. The wounds that are frequently followed by malignant œdema are punctured wounds of the muscles, such as those caused by a stake or by a stable fork. Wide, open surfaces are less frequently the seat of this disease.

The prevention of the disease in surgical wounds is, of course, the observation, as far as it is possible in veterinary practice, of aseptic surgery. The curative treatment will depend for its chance of success upon the length of time the disease has been established and its extent. The object to be sought is to open the lesion up to the air to as great an extent as possible—amputation is not as a rule applicable to animals—by cutting into it, then irrigating it out with an antiseptic solution—peroxide of hydrogen being indicated—getting a thorough drainage from the deeper parts established, then dressing it with iodoform or tincture of iodine.

At the same time the patient should be kept up with stimulants and tonics.

In Sheep.

It has always been recognised as a fact in farming practice that the operation of castration in sheep is attended with greater risk than it is in cattle, also that parturition in these animals leads to more deaths than is the case in cattle. In both instances many of the cases that occur are diagnosed as "blood-poisoning," and undoubtedly a number of such cases are due to the presence of the malignant œdema bacillus. The freedom of cattle from such complications is due to the fact that cattle are not susceptible to the disease.

Oppermann, in 1913, published a lengthy article upon this subject. He commences by recalling the fact that malignant œdema complicates by preference wounds of the skin and mucous membranes which are necrotic, which bleed little, and the edges of which, after a short time, close and become scabbed, thus impeding the access of air—that is, of oxygen—which is the chief bactericide against anaerobic germs. The practically constant presence of the specific microbe of malignant œdema in most solid materials which come into contact with the animal body—the superficial earth, air, litter, instruments of grooming and of shearing, walls of stables, fences, more or less dirty hands and clothing of attendants, etc.—makes it possible for the disease to appear upon all farms. Moreover, it is now known that the receptivity of the animals of the farm is general. The horse, rabbit, pigeon, guinea-pig, and mouse may all be naturally infected, and nearly always succumb to the disease. The reason why malignant œdema is not observed daily is that usually phagocytosis (protective action by the white corpuscles of the blood) is so active in the parts inoculated that the spores introduced

into the tissues are unable to develop to bacilli. This phagocytosis may be counteracted or annihilated by the accidental presence of other germs, such as staphylococci, the *Bacillus prodigiosus*, etc. In these cases, the disease generally follows its normal course. It is also known that whenever the tissues are infiltrated, severely contused, or necrosed, the spores of malignant œdema may evolve to the bacterial form without needing the co-operation of microbes neutralising phagocytosis.

In sheep, malignant œdema is a mortal affection. The author names four principal accidental causes—namely, parturition, castration, shearing, and bites of dogs.

The treatment of the disease does not offer much hope of success. It is similar to that recommended for horses, and consists of lancing the swelling and the injection of a 2 per cent. solution of peroxide of hydrogen or permanganate of potassium.

The preventive treatment consists of disinfection of the lambing quarters, and other antiseptic precautions as far as possible, and aseptic surgery.

In Pigs.

Cases of malignant œdema are sometimes observed in the pig after castration, but in my experience they are of rare occurrence.

ACTINOMYCOSIS

Synonyms.—Lumpy Jaw, Wooden Tongue, Ray Fungus Disease.

History.—The ray fungus has been known of since the middle of the last century, but it was not until 1877 that Bollinger, in his work on actinomycosis of the jaw of cattle, demonstrated the real significance of the disease. The disease is widely spread, being very common in Russia, the Western States of America and the Nile Valley. It is of less frequent occurrence in Denmark, Holland, England, and Germany.

The disease was observed in Natal about the year 1900 by J. L. Webb, F.R.C.V.S., and in 1911 he reported that the disease was becoming prevalent in the midlands of that province amongst graded and pure-bred cattle. He was of the opinion that the disease was introduced by imported bulls, which infected the farms, and that the disease subsequently spread from such centres. The writer's experience of the disease in Rhodesia has been limited to a small number of cases that have occurred

in imported cattle, but the disease has not been observed in locally-bred animals.

Nature.—Actinomycosis is caused by the ray fungus, an organism that was supposed to belong to the higher fungi, and to be closely related to the moulds. This idea no longer obtains, and the organism is now recognised as a streptothrix—*i.e.*, a bacterium of which the filaments exhibit genuine branches.

The actinomyces (ray fungus) is a facultative parasite. Its normal method of existence is to grow as a parasite on vegetable structures, especially in the cereals, particularly on barley. Circumstances under which the disease occurs also appear to show that the organism may be present on some of the grasses. Actinomyces have been demonstrated on vegetable structures.

The disease is practically always sporadic, and exhibits no tendency to spread by contagion. When a number of animals are simultaneously affected, the cause must be put down to them having derived the infection from the same source.

The actinomyces is pre-eminently a tissue parasite. It has never been observed to have a septicæmic tendency; it never multiplies in the blood, and it has never been seen there, although it appears in some cases to be distributed by the blood-stream. Broadly speaking, it is confined to the lesions and their discharges.

The animal most commonly infected is the ox, but cases of actinomycosis are met with in man, the horse, sheep, pig, and dog. It appears to have been established that the majority of cases met with in man are identical with cases found in cattle, and the actinomyces found in the human subject is not distinguishable from the same organism found in cattle; but no case of infection from animals to man of this disease has been recorded, either by meat, milk, or inoculation. The habit of sucking straws is regarded as a source of infection in man. It is difficult to transmit the disease experimentally, even from one ox to another; and when it has been successfully done, the resulting lesions are very restricted and non-progressive, and after a short time they become retrogressive. Even subcutaneous inoculation with a number of organisms sometimes fails to produce the disease.

Natural infection in the immense majority of cases takes place through the mucous membrane of the mouth. It is an open question if it happens if the membrane is intact, but the pricks of the ends of the forage may perhaps lacerate the membrane.

The primary seat of the disease is often in the vicinity of the

molar teeth. Shedding of the milk teeth is a predisposing cause of infection. What is apparently primary infection is occasionally met with in the udder of the cow and the sow, the route of the infection having apparently been by way of the teat canal. Wound infection also occurs. This is seen in cases of schirrous cord in cattle.

Symptoms.—There is no constitutional disturbance, and there are no symptoms except those of a mechanical nature, owing to the situation of the lesion interfering with the process of nutrition. By far the commonest seats of the disease are the mucous membrane of the mouth, especially the tongue and the bones of the upper and lower jaws. Next in frequency come the glands of the throat and between the jaws, and the mucous membrane of the throat. Lesions are frequently found in the nasal mucous membrane, but as a rule they are only extensions of the mouth lesions. When the site of infection is the jaw, the name "lumpy jaw" is given to it; when the tongue, "wooden tongue"; and if the soft part of the throat, it is called a "wen or clyer." The disease is always of a chronic character, and it rarely extends beyond the lymphatic glands of the throat. Abdominal lesions by metastasis are extremely rare, but they are met with in the lungs and liver. Webb states that in South Africa he has never seen any parts other than the jaw bone affected. The cases I have seen in Rhodesia have also all been in the jaw.

Tongue Lesions.—At the time when the disease is clinically recognisable, this organ is more or less enlarged and indurated, and any part of, or the whole of, the organ may be so affected. The enlargement is sometimes so great that the tongue protrudes from the mouth, and when the lower half is affected the tongue is often distorted as well.

Ulceration of the mucous membrane occurs quite early in the disease. It is quite shallow, and only extends the depth of the mucous membrane. These patches may be circular or jagged in outline, and look as if the mucous membrane had been etched away with acid.

On cutting into the tongue, there is found at places under the mucous membrane to exist a quantity of reddish-grey, glandular new tissue, always very rich in colonies of the organism. By scraping the surface and smearing it on a glass slide, and then holding up to the light, they can be detected easily.

The cirrhosis of the indurated parts of the tongue is due to fibrous tissue that is formed at the expense of the muscular tissue, the size of a grain of barley, firm, and not readily enucleated.

Suppurative lesions in the tongue are exceedingly rare.

Lesions in Bones.—These are practically never primary, but are mostly secondary, to lesions in the alveoli (teeth sockets) or hard palate. It is a destructive ostitis (inflammation of bone), but at the periphery of the lesion a good deal of new bone is formed, and the diseased area becomes greatly extended by this means. The disease extends to the soft structures of the face, and bursts outwards, discharging a thick yellow pus very rich in colonies of the organism.

Lesions in the Glands.—When the disease affects the pharyngeal or parotideal (throat) lymphatic glands, it often leads to an enlargement that encroaches upon the pharynx (throat) and interferes with the respiration. Polypus-like growths from the mucous membrane of the throat sometimes occur; these also interfere with respiration.

Sometimes the glands burst outwards. In this position the lesions of actinomycosis have the most resemblance to tuberculosis, and a microscopical examination is often necessary to decide which disease is the cause of the lesion. Where pus is obtainable, there is little difficulty in diagnosis by microscopical examination. The organism, like all other known streptothrices, shows a marked tendency to colony formation in tissue. Some colonies are easily visible to the naked eye. If pus is spread out on glass and held up to the light, they may attain the size of a small pin's head. They may also be felt when pus is rubbed between the fingers, but as a rule they are only just visible as mere specks.

Other seats of the disease are the spermatic cord in castrated animals and the udder in the cow (see Mammitis).

Treatment.—Prior to the iodine treatment for this disease, recommended by Thomassen in 1885, becoming generally known to veterinarians, the methods, or at least some of them, adopted for the cure of this disease were somewhat crude and barbarous; one of which, that I had good results from, was to paint the tumour with a mixture composed of equal parts of turpentine and sulphuric acid. This is a dangerous mixture to prepare, and I do not advise anyone to mix these ingredients together. After dressing the "wen" and upon releasing the poor brute, it would gallop off with the hot air plainly visible coming from its nostrils. Other practitioners used nitric acid for a similar purpose, and also claimed good results from it.

The other method of treatment adopted was a surgical operation to extirpate the tumour. Probably even at the present day, in the hands of a capable operator, this is the most certain method

of dealing with those cases in which the tumour is sharply circumscribed, surrounded by healthy tissue, and situated in a site that lends itself to the operation. Schirrous cord falls within this category, but many cases are of such a nature that a surgical operation is impossible, and in these iodide of potassium, which is a specific for the disease in cattle, should be employed.

The dose should be regulated according to the size of the patient, about ¼ dram being given for each 100 pounds of body weight—that is, 2 or 3 drams given daily in a pint of cold water as a drench, and repeated daily for a month or six weeks. After the drug has been administered for some time, symptoms of iodism may appear in some animals in the form of loss of appetite, a watery discharge from the eyes and nose, the fæces become dry and perhaps coated with mucus, and the skin becomes scurfy. In such cases the drug must be stopped for two or three days. In some cases it may be necessary to continue the treatment for longer than six weeks. Perhaps the treatment gives its best results in cases where the tongue is affected, and in all cases where it is adopted early in the disease, and is not so effective where the jaw bone is involved, but I have had recoveries in cases where the jaw bone has been seriously affected. A treatment that has been advised in place of the foregoing is biniodide of mercury, 3 or 4 grains, with 10 grains of iodide of potassium in a pint of water daily, and repeated for a similar length of time, but in my experience the results are not so satisfactory.

One of the greatest objections to the iodide of potassium treatment is its cost, but (in South Africa), owing to the patient being frequently a high-priced imported bull, a trial is often justified. In this connection another action of the drug must also be taken into consideration. When it is used for long periods it not only produces emaciation, but it may also cause atrophy of the glands; and if this should happen to the testicles, there is the probability of the bull becoming sterile. To counteract these unfavourable results following, the treatment should not be persisted in for longer than necessary, and the patient, when undergoing it, should be supplied with plenty of nutritious and easily-digested food. In the case of cows in milk undergoing treatment, some of the drug is excreted by the mammary gland, and renders the milk unfit for use and for the rearing of a valuable calf. In treating cases where suppuration is present, the ordinary antiseptic precautions and dressings should be applied. After syringing the abscess out, it may be dressed with tincture of iodide,

or a pledget of wool saturated in iodised phenol may be plugged into the orifice. In cases affecting the jaw bone it is well to rub in the ordinary red blister (biniodide of mercury, B.P.), and in cases of the tongue some veterinarians scarify this organ first and then rub the foregoing blister on; but such treatment as the latter is perhaps unnecessary and unjustified.

Good results have been claimed for a treatment in which iodide of potassium is injected in solution direct into the tumour twice a week, instead of giving it as a drench.

BOTRYOMYCOSIS

Synonym.—Discomycosis Equi.

History.—Bollinger was the first to observe the cause of the disease in the nodules of a horse's tongue (1869); subsequently it was observed by Rivolta and Micellone (1879), Johne (1884). and by Rabe (1896). Since then the disease has been observed by numerous investigators.

Nature.—The disease is caused by an organism known as the botryomyces, which is sometimes called the discomyces or *Staphylococcus ascoformans*, which greatly resembles the *S. pyogenes aureus;* in fact, Sir John MacFadyean states: "Although a contrary opinion has been expressed by some observers, apparently it must now be admitted that the so-called botryococci, which can be cultivated from any case of botryomycosis, are indistinguishable from the common *S. pyogenes aureus* by cultural, morphological, or staining characteristics."

Like the *S. pyogenes aureus*, the botryomyces is ubiquitous in nature. It is only as it is found in chronic lesions that it differs morphologically from the *S. pyogenes aureus*. In such lesions there are always present diplococci and staphylococci in groups the same as in lesions of *S. pyogenes aureus*, but, in addition, botryomyces granules are constantly found. These are, in reality, colonies of staphylococci, and, like the actinomyces granules, they are visible to the naked eye, and under a low magnification they might be mistaken for actinomyces colonies. The colony presents a composite character, roughly spherical in shape, compacted of a variable number of spherical masses adhering together. Its outer edge has a scalloped appearance, but never any appearance of toothing, such as is caused by the clubs in the actinomyces colony. It has been held that the cause or the meaning of the colonies is a successful attempt on the part of the tissue to resist the organism. In support of this theory, it is

pointed out that colonies have never been found in artificial cultures, nor are they found in acute lesions.

Although these two organisms are so similar in many respects, there are differences between them. When a culture of *S. pyogenes aureus* is injected under the skin of a horse, it provokes the development of a boil-like lesion, intensely sensitive and painful. Within two or three days this softens and bursts spontaneously, and discharges a thick, creamy pus, and after discharging for a few days the abscess dries up and disappears. When a botryomyces culture is similarly employed, the result may be identical, but sometimes the abscess leaves a lesion under the skin, and there forms then a good deal of fibrous connective tissue; and after weeks or months the abscess will burst spontaneously again, and the pus will contain the characteristic granules. The *S. pyogenes aureus* never does this.

In the case of acute abscess, it is not possible to say whether the staphylococci found to be present are the *S. pyogenes aureus* or the botryomyces.

The disease is a sporadic affection that usually occurs in isolated cases which show no tendency to spread. It is almost exclusively found in the horse, but cases have been found in cattle and in man. In the horse the methods of infection are the same as in the case of *S. pyogenes aureus*. Harness is regarded as a frequent means of transmitting the disease from one animal to another. The commonest lesions produced by the disease are schirrous cord, the shoulder tumour at the point of the shoulder (the elbow tumour, "capped elbow," less frequently becomes infected), fistulous withers, and poll evil. A tumour-like growth appears sometimes on the stump of the tail after docking. The udder in the mare is another seat of the disease. Metastatic lesions in the internal organs are not very common, but are met with in the spleen and lungs, often secondary to a large schirrous cord. In the depth of a schirrous cord or shoulder tumour, the botryomyces are found as a pure culture, but in poll evil and more open lesions a mixture of bacteria is usually present.

Symptoms.—The symptoms are usually limited to the lesions, and as these are, in the majority of cases, situated in places where none of the vital functions of the body are interfered with, few or no constitutional disturbances, such as loss of appetite or emaciation, are observed. Exception must be made in this connection in the case of schirrous cord, which forms in connection with the stump of the spermatic cord after castration. It may attain to 7 or 8 pounds in weight, causing

considerable pain and lameness, which naturally leads to loss of condition.

All botryomycotic lesions, when superficial, have the same characters. They lead to the production of a tumour-like mass of dense, inflammatory, fibrous tissue, and small abscesses form in connection with the skin over the tumour. These burst spontaneously. They may heal up, but fresh abscesses continue to develop from time to time.

When a schirrous cord or a botryomycotic tumour which has been excised is divided with a knife, it is found to be composed of a dense, hard substance, which is really coarse, fibrous, connective tissue, and scattered through this substance are soft centres and abscesses from which fistulæ may lead to the exterior. The pus may be yellow and creamy, but frequently it is somewhat mucoid in character, and sometimes it is brownish in colour. It always contains the granules.

Treatment.—The treatment adopted is similar to that employed in actinomycosis, only that the knife is used more frequently.

In all cases, where possible, the total excision of the growth should be attempted. Some operators claim that if only the major portion is removed and the remaining portion treated with tincture of iodine the growth will disappear, and whilst this is undoubtedly true in many cases, it is better to attempt to make a "clean job" of it. In dissecting out a growth, considerable hæmorrhage usually occurs, necessitating ligature of the blood-vessels as one proceeds with the operation.

The treatment for botryomycotic lesions is tincture of iodine or biniodide of mercury blister, which is applied both to the surface of the growth and into its substance by way of the abscess cavities.

A diversity of opinion as to the efficacy of the internal administration of potassium iodide exists. Whilst a number of practitioners report favourably of its effects, an equal number report failure. The conclusion that perhaps should be drawn is that the reliance placed upon this drug in the treatment of actinomycosis cannot be accorded it in botryomycosis; but failing other methods of successfully combating the disease, or in cases that are caught in the early stages, a 2-dram dose of iodide of potassium should be given once or twice daily to a horse.

NAVEL ILL

Synonyms.—Joint Ill, Pyæmic Umbilical Infection, Specific Synovitis, Specific Polyarthritis.

History.—The disease was first described in 1781 by Brügnone of Turin, and subsequently by other observers; but it was not until Bollinger studied the disease in 1873-1875 that its true nature was recognised by him. In later years the disease has been investigated in England by Penberthy and others.

Nature.—The nature of the disease is a pyæmic infection of the umbilical cord, causing lesions in various parts and organs of the body, but particularly in the joints, especially of foals, and less frequently in calves, lambs, and pigs. Some uncertainty still exists as to the organisms causing the disease. The *Staphylococcus pyogenes aureus* and the *Streptococcus pyogenes* have been implicated, also the necrosis bacillus. Other authorities assert that an organism of the fowl cholera type is the causal agent in many outbreaks of the disease, and whether the infection is a single or mixed one is still in doubt.

The infection takes place at, or shortly after, birth through the unclosed navel, but cases have been recorded in the foal within the womb, and Penberthy observed that many such foals were born prematurely. The first symptoms of the disease are usually seen when the foal is from three days to three weeks old, but cases, usually of a subacute nature, occur when the foal is a few months old. More cases are seen in colts than in filly foals, which may be explained by the discharge of the urine keeping the navel moist and open, and favourable to the growth of organisms. The course of the disease is generally acute, death occurring in a few days. The average duration is said to be about two weeks. The general rule is, the older the foal the longer the disease lasts.

The disease is more prevalent in some countries than in others, and in one part of a country than in other parts. Once the disease has become established in a stud, extraordinary precautions have to be taken to prevent its annual recurrence. The mortality is anything from 40 to 60 per cent. or higher, and usually a foal that has recovered from the disease is a pretty worthless animal. Of course, cases do occur where no appreciable after-effects are seen.

The common method of infection is by means of the blood-clot that is usually present soon after birth in the umbilical cord at the navel. Inflammation of the umbilical vessels, especially

the vein, is first set up, which is frequently followed by a breaking-up of the clot inside the navel, resulting in metastatic pyæmia of various structures, particularly the liver and lungs, but the synovial membranes of the joints are almost invariably attacked.

Symptoms.—The primary symptoms are those of systematic disturbance. The foal is dull, lies down, disinclined to move or suck, the temperature 104° to 106° F., and breathing quickly. These symptoms may exist for one or two days before the first really typical symptoms—a swelling of one or more joints accompanied by lameness—appear, or this sudden swelling and lameness may be the first thing to be seen.

Any joint may be attacked, but perhaps the stifle or hock joints are more frequently attacked than the others. The swellings are hot, hard, and painful, and may be, and frequently are, mistaken for injuries or rheumatism. The constitutional symptoms increase in severity, and abscesses form in and around the joints; or the hair may fall off, and a yellow fluid exude from the swelling, which in turn is followed by the skin sloughing off, exposing the ligaments and tendons.

At the commencement of the disease, if the navel is examined, it will be frequently found to be swollen, moist, or weeping, and unhealthy in appearance. Diarrhœa is a fairly constant symptom, frequently of a fœtid or offensive nature. The other symptoms shown will vary according to the organs and tissues implicated in the disease; inflammation of the lungs, pleura, peritoneum, or heart, etc., may be present.

Treatment.—The treatment to be aimed at is one of prevention, and consists in thorough cleansing and disinfection of stables and boxes in which the disease has occurred, and isolation of infected animals and their special attendants.

The disinfection should also include that of the mare before foaling by washing the hind parts, thighs, and tail with a disinfectant solution; and an attendant should be constantly on the watch armed with materials for ligaturing the cord and dressing the stump directly the foal is born.

The ligature may be surgical silk or soft twine that has been soaked in a corrosive sublimate solution (1 to 500) previously, and should be tied about an inch below the navel ring, and the cord cut through about ½ inch below the ligature. To ensure success, the ligature should be applied directly after the foal is born before the navel cord can have become contaminated with organisms It is not advisable to ligature if the foal has been born some time, as it only imprisons the organisms, and if

the cord has been broken, it is often difficult to apply the ligature.

The dressings employed are corrosive sublimate (1 in 500). This is used for washing the end of the cord and its surroundings after it has been ligatured. It is then dried, and the stump of the cord is painted with pure carbolic acid. This painting is repeated daily for a few days until the part has become quite dry and healed. Other practitioners prefer the use of tincture of iodine to carbolic acid. The treatment is varied according to the nature of the case, and to meet to a greater extent the varying conditions the following dressing is largely employed. This is a solution of salicylic acid in collodion (1 in 10). The stump of the cord is first sponged with alcohol, or it may be dressed with the corrosive sublimate solution and dried thoroughly, then painted with the collodion, which is repeated daily until the navel dries up.

If the cord has broken off short, the same treatment may be applied, or the part may be dressed with tincture of iodine and the corrosive sublimate solution.

The disease, when it occurs in calves and lambs, is very similar to that described in foals, and the treatment is the same.

Various forms of medicinal treatment have been adopted for affected animals, but with indifferent success. When suppuration occurs in the joints, the case is as a rule hopeless.

No specific treatment is known, and each case has to be treated on its merits. Good nursing, opening of abscesses, disinfection, and tincture of iodine are stock treatments. Marked success is said to have followed the injection of 2 to 4 drams of a solution of acetozone (1 in 500) into different parts of the affected joint, coupled with the administration every five hours of a tablespoonful of the solution mixed in 2 ounces of the mare's milk.

VACCINE THERAPY

VACCINE therapy, or the treatment of disease by vaccines, to a greater extent for curative purposes than vaccines have been hitherto employed, has been attended with very successful results in human practice, particularly in the treatment of acne and other lesions caused by the *S. pyogenes aureus*. This method of treatment of diseases, particularly those of a suppurative nature, has been introduced into veterinary practice, and was used to a considerable extent amongst the animals in the Expeditionary Force of the British Army in France in the recent Great War.

Vaccine treatment is said to succeed best in strictly localised and chronic infections in vascular tissues, and to a less extent in subacute conditions. It has been found beneficial in cases of simple abscess, multiple abscess, suppurations, ulcerations, strangles, mammitis, catarrh, pneumonia, etc. In addition to the foregoing, the treatment has been applied to more acute and serious bacterial diseases, perhaps, up to date, with less successful results; but it must be remembered that the method, at least in some of the diseases, is in its infancy, and better results may be possibly anticipated in the future.

The vaccines employed are classified as "stock" vaccines and "autogenous" vaccines, and are prepared from the artificial cultivation of the causal and associated organisms.

Stock Vaccines.

Stock vaccines are prepared from cultures of those organisms which experience has taught are commonly associated with the diseased condition, such as the streptococcus in strangles.

Autogenous Vaccines.

Autogenous vaccines are prepared directly from cultures of those pathogenic organisms which are found by bacteriological methods to be actually present in the diseased tissues of the particular case in question.

Autogenous vaccines should possess many advantages over stock vaccines, for the reason that so many cases in animals are mixed infections. Of course, even with autogenous vaccines there is the risk that the chief organism in a mixed infection may have been crowded out by the others; or it may not be collected in the material taken from the lesion for cultivation; or, further, that it may fail to grow in the medium in which it is sown.

These vaccines consist of quantities of killed organisms of the same species that cause the disease, or that are associated with the diseased conditions. When vaccines contain several species of organisms found in the diseased lesions they are spoken of as polyvalent vaccines. Stock or autogenous vaccines may or may not be polyvalent.

The Principles of Vaccine Therapy.

In the first place, it has always been held, and I think correctly, that an attack of infective disease confers an immunity to a second attack—what we know in this country as "salting." This immunity may be for life or lasting, or it may be of very short duration. The reason for this immunity is attributed to the presence of immune bodies, or "antibodies," as they are more frequently called, in the blood and tissues. The exact nature of these bodies is unknown. They comprise the antitoxins, agglutinins, opsonins, etc., of the bacteriologist.

It is the healthy tissues which produce the antibodies, and these are carried by the blood-stream to all parts, including the infected sites, where their action is supposed to be that of rendering the bacteria in some unexplained way more attractive to the leucocytes (white blood-corpuscles), which envelop and destroy them.

Sir A. E. Wright has shown that this treatment has a marked effect in increasing the opsonic (antibody) index of the blood, and he has aptly described the treatment as an exploitation of the healthy tissues for the benefit of the diseased.

The vaccine is employed as a subcutaneous injection. In the Expeditionary Force in the Great War vaccines were also used as wound dressings, and it has been found that the immediate result of injection is that for the first twelve to twenty-four hours there is a diminution of the number of antibodies present in the blood. This is known as the negative phase, and is followed by the positive phase, in which the antibodies are increased in number. The object to be arrived at is to get a slight negative phase, followed by a good positive phase.

In the Expeditionary Force it was customary to put the patients through a course of treatment. The first injection consisted of 1 c.c. of the vaccine. After two or three days had been allowed to elapse 2 c.c. were injected. The dose was increased by 1 c.c., and repeated at similar intervals until 5 c.c. were reached, when the course was complete, but might be repeated again in a week if necessary.

The Effects of the Vaccine.

During the negative phase there may occur a rise in temperature of 1° to 1·5° F., the pulse and breathing become slightly quickened, the clinical symptoms at the site of the disease may be increased, with a slight general discomfort of the patient.

During the positive phase, the temperature falls to where it originally stood, or lower, or to normal, and a general return to, or improvement in, the conditions prior to injection.

Of course, during the vaccine treatment the other treatments recommended, such as the dressing of wounds and attention to the general health and comfort of the patient, are still pursued.

BACILLARY NECROSIS

Synonym.—Necrobacillosis.

Definition.—Necrosis means the death of tissue, and bacillary necrosis, or the death of tissue due to a particular bacillus, has been proved to occur in a number of diseases.

History.—The discovery of the bacillus of necrosis was made by Löffler in 1884, when investigating the disease known as "calf diphtheria," and was attributed by him to be the cause of that disease. Since that time the presence of the bacillus has been demonstrated by other observers as the sole or principal causal organism of the lesions in several other diseases. In South Africa it has been found in various lesions in different species of animals.

Nature.—The necrosis bacillus, or the *Bacillus necrophorus*, as it is sometimes called, is a facultative parasite which is widely distributed in the soil, and it is a very common, if not constant, inhabitant of the alimentary canal of different animals, especially the herbivora. The disease caused by it occurs sporadically, and with regard to its contagiousness a difference of opinion exists, the same as it does with some other sporadic diseases. There appears to be little doubt but that a form of the disease that occurs in rabbits is contagious, but, without giving an opinion one way or the other, the wisdom of treating all cases of the disease as being contagious is recognised by all.

The necrosis bacillus is essentially a tissue parasite confined to the lesions. It is never septicæmic, but occasionally it is metastatic, being transported either by the blood or the lymph stream, but most frequently by the former. In recent lesions the bacilli are very numerous; in old lesions they are very scanty and difficult to demonstrate by the microscope.

The bacilli appear to be pathogenic for a great variety of animals, and are responsible for lesions in all domesticated mammals, and occasionally in man himself. The disease is not very readily experimentally transmitted unless large quantities of infective material are used.

Natural infection in a good many cases takes place through

wounds, but it may take place through intact mucous membranes.

Amongst the animals susceptible to the organism, and the diseases in which the necrosis bacillus is the actual cause or a serious complication, are the following:

In Cattle.

Calf Diphtheria.—This is the most important and the commonest form in which disease caused by the necrosis bacillus is met with in cattle. The disease was recognised for many years in Germany as a calf disease before its cause was known. In England it was described as malignant stomatitis. Löffler investigated the disease in Germany in 1884. A little later Bang investigated the disease in Denmark, and gave the first description of the organism. Consequently it is frequently called the necrosis bacillus of Bang.

It is often a formidable disease of quite young calves, and large numbers are frequently affected in quick succession.

The lesions involve the tongue and the mucous membrane covering the jaws and cheeks; sometimes also the soft palate, the throat, and the larynx.

Symptoms.—The affected calves lose their spirits, cease to suck, salivate, and show much difficulty in swallowing. On examining the animal closer, a hard, painful swelling may be found on one or other cheek. Opening of the mouth to examine it gives the animal pain; the tongue is swollen and its movements limited. Grey patches of various dimensions and thickness of a yellow deposit are found on the tongue, gums, or cheeks. These deposits are readily detachable. The animals are fevered, and diarrhœa frequently supervenes. A little later a discharge from the nose may take place of a yellowish colour that tends to block the nostrils and cause difficulty in breathing. A cough is usually an indication that the throat or the lungs, as they frequently do, have become affected.

Extensive lesions in the mouth are usually rapidly fatal (within a few days), but when the lesions are superficial and of smaller extent the deposit may be cast off, leaving deep ulcers, and the animal may linger on for some weeks, extension of the disease to the lungs frequently terminating the case. When the lesions are confined to the tissue of the cheek, they may persist for a long time, becoming incapsulated, and eventually becoming completely absorbed.

The disease is commonly met with in the Union and Rhodesia as sporadic outbreaks in which frequently only one calf is affected, but that the disease can assume much more serious proportions was shown in an outbreak that occurred in Bechuanaland in 1911, which was investigated by Mr. W. H. Chase, P.V.O., who described the symptoms as follows: "The calf starts passing a frothy, yellowish purge, refuses to suck, and the coat stares. Blood-stained saliva dribbles sometimes; the nasal discharge is also blood-stained. Respiration is accompanied by grunting and sometimes by a cough. This is the general run of the cases. There are a few recoveries, but not many."

The outbreak occurred on three adjoining farms in the Lobatsi block, and affected calves up to twelve months old. In one case lesions of the udder were noticed, but in this instance the animal recovered. On one farm there were 100 calves under twelve months old. Of these, 65 died. On another farm of 17 animals affected only 5 recovered. Mr. Chase stated that the foregoing is about the average percentage of recoveries.

Treatment.—The main treatment consists in arresting the spread of the disease. The healthy calves should be moved into new quarters if possible, and the old calf quarters should be thoroughly disinfected, as well as everything that has been in contact with the diseased calves. Perhaps, through force of circumstances, the healthy calves will have to remain in the quarters and the sick ones be removed and isolated. If so, the disinfection should be very thorough, particularly with regard to the floor. If this is of soil, it should be dug up and a fresh floor put down.

Care should be taken that attendants' hands are disinfected. In hand-reared calves the feeding utensils—particularly the bucket—largely spreads the disease.

With regard to medicinal treatment but little can be done. The mouth should be washed out and the necrotic tissue removed, and the sores painted with a weak solution of permanganate of potash. The calf must be well nursed, and every endeavour made to keep its strength up.

Foul in the Foot.—Foul in the foot, or Klawziekte, is another disease in cattle in which the necrosis bacillus plays an important part by causing the sloughing of the tissue between the digits. Unless attended to very early in the attack, this is a very troublesome complaint to deal with. It causes great lameness, pain, and loss of condition, which may end in death. To save an animal's life, amputation of the limb above the fetlock-joint has been practised.

The treatment consists in hot fomentations; soaking the foot, if the animal will permit it, in a bucket of hot water; removing the ragged edges of the horn and the dead tissue from between the claws; and treatment of the sore with antiseptics. Strips of calico soaked in carbolised oil and drawn up between the claws help greatly. At the commencement it is often advisable to put the foot into a small sack containing a hot bran poultice for the purpose of separating the dead tissue from the healthy. Great benefit follows, but it must be remembered that the necrosis bacillus is an anaerobe, and cannot grow in the presence of oxygen. Therefore, by keeping the part constantly clogged up with poultice, and so excluding the air, more harm than good may result. The object to be aimed at is the removal of dead tissue, and allowing the air and antiseptic dressings to penetrate to all parts of the lesion.

Navel Ill.—The necrosis bacillus is also responsible for some of the cases of so-called navel ill in calves. The organisms enter the body by the uncicatrised umbilical cord. The post-mortem lesions are present in the liver in the form of circumscribed tumour-like nodules, and occasionally similar nodules are found in the lung tissue.

Treatment.—See Calf Diphtheria and Navel Ill (Pyogenic).

Liver Lesions.—Precisely similar hepatic nodules are not rarely found in adult cattle, and in most of the recorded cases the liver lesions are the only ones detected; but probably the primary lesions are in connection with the stomach or intestines, and extensive lesions of the walls of any of the first three stomachs are frequently met with when they are looked for.

The liver is usually greatly enlarged, and the nodules may be as big as a walnut. In consequence of the well-defined, rounded form, firm consistence, and the sharp demarcation, they may be mistaken for neoplasms (tumours). When they are quite recent they have a firm, dry consistence, and they cut with a smooth surface. Sections with no tendency to disintegrate may be cut from them, thus distinguishing them from tuberculosis. They are dirty-white in colour, and one can obscurely trace the lobulation of the liver in them. The edge of the lesion is generally sinuous, and between the light-coloured necrotic part and the brown liver tissue there is a narrow dark stratum that contains the filaments of the necrosis bacillus in great abundance.

Bacillary necrosis lesions have been found in the lungs and liver of calves, and in the stomach, peritoneum, and liver of adult cattle in South Africa.

In the Sheep.

Lambs are also subject to navel infection with the necrosis bacillus, and the lesions are identical with those present in the disease in calves. The disease is very fatal, and a frequent cause of extensive loss.

Another route of infection for the organism is through the docked stump of the tail. In such cases the stump is usually thickened, and shows an ulcerating surface. In 1910 the disease was diagnosed in a stud flock of merinos at Amersfoort, near Volksrust, causing great mortality. At the time of diagnosis about forty lambs were dead and many more were ill. The main symptoms noted were an affection of the mouth, showing inflammation of the mucous membrane and a swelling of the joints, which sometimes burst and discharged pus. A contagious form of foot rot is caused by the bacillus.

Treatment.—See Navel Ill (Pyogenic) and Calf Diphtheria.

In the Horse.

The necrosis bacillus is largely responsible for quittor, but all quittor lesions contain a considerable mixture of bacteria.

In cases of poll evil, fistulous withers, and similar chronic suppurating conditions associated with necrosis of some of the deeper tissues, which have previously been attributed solely to the action of pyogenic organisms, there is no doubt present now but that the necrosis bacillus plays a most important part in many such cases.

The organism is also responsible for sloughs in horses' heels and at the top of the hoof, where no injury is present; it is also present in pricks in shoeing and in gathered nails. In any of these conditions about the foot, metastasis may occur by the veins, and necrotic foci occur in the lung tissue. In a good many such cases the secondary lesions, like the primary, contain a mixture of bacilli, but the necrosis bacillus is the chief offender. The pleural lesions may also be a secondary infection from the pulmonary one.

The necrosis bacillus is also responsible for an ulcerated condition of the horse's intestines that resembles the ulcers of swine fever.

Fortunately the foregoing lesions—quittor, poll evil, fistulous withers, etc.—are rarely met with in South Africa.

In the Pig.

Bang maintained that the necrosis bacillus is sometimes mainly or entirely responsible for the deeper lesions (*i.e.*, the ulcers) found in the intestines in swine fever. Other authorities regard the organism in these cases as a secondary infection.

In America there is a serious form of the disease in the pig commonly termed "snout disease," "bullnose," or necrotic rhinitis, which is incurable and often associated with rickets.

PURPURA HÆMORRHAGICA

Synonym.—Petechial Fever.

History.—The disease has been known for a long time, and various views have been held as to its nature. The disease occurs throughout South Africa, but it is not so frequently met with as in Great Britain, nor, as a rule, are the cases so severe.

Nature.—The disease is commonly regarded by authorities as a microbic affection, but the causal organism has not yet been determined, and attempts to transmit the disease from horse to horse have failed.

It is rarely, if ever, the disease occurs as a primary affection. In nearly all cases it follows some other trouble. Strangles and influenza precede it in, perhaps, the majority of instances, but it may follow lung trouble, or even a wound. It is not essential that the patient should have suffered from one of these diseases severely, although it is so in most cases, but occasionally purpura hæmorrhagica follows a mild attack of strangles or influenza.

The belief is held by some authorities that the typical symptoms of the disease, the swellings and the blood-spots on the mucous membranes, are the result of the poisons (toxins) formed by the micro-organisms.

The disease never creates the impression of being directly contagious, but when one case of it occurs in a large stable it is frequently followed by others. Unsanitary conditions, such as ill-ventilated, badly-drained, dark stables, and debilitating causes predispose animals to the disease.

Various organisms have been found in connection with the disease. The different staphylococci are frequently found, but further investigations are necessary to determine the actual cause of the disease.

Symptoms.—These, as a rule, appear suddenly, and consist of swellings about the head, the limbs, the abdomen, or the chest, and of small purple spots (petechiæ), like flea-bites, on the mucous membrane lining the eyelids and nostrils. The swellings, when they first appear, may be very much like those seen in nettlerash (urticaria), and they may move from one part of the body to another. For example, they may move from the hind to the

front legs, or to the head, or the movement may be in the opposite direction. The foregoing are the commonest sites for the swellings, but they may appear on the under-surface of the abdomen and sheath, and a similar extravasation may occur internally, when acute colicky pains are manifested, which are generally followed by death.

The swellings are usually not very hot or painful; they pit on pressure, and terminate abruptly. Those on the limbs look as if a cord had been fastened tightly around the limb, and the swelling had occurred below the ligature; on other parts of the body the termination of the swelling is also abrupt, but not so marked as in the case of a limb. In the latter position, the swellings sometimes assume enormous dimensions, and a yellowish fluid oozes out. In such cases the skin is liable to crack in places and pieces of it slough off, leaving very nasty sores.

The swellings about the head, frequently at the commencement, appear about the lips and nostrils. Later on the whole head may swell up to such a size that the animal has great difficulty in breathing, and tracheotomy (tube in windpipe) has to be performed to save the patient's life. This operation should always be avoided if possible in this disease. Such swellings, according to their extent, always make it more or less difficult for the animal to eat. At times the only nourishment that can be swallowed is thin gruel, and only then if the animal can get its head deep down in the bucket.

The foregoing are the typical symptoms of the disease. They are frequently accompanied by a soft cough, and generally by a bloody-coloured mucous discharge from the nostrils.

The petechiæ will at first appear like flea-bites. They tend to run together, and form irregular patches of various sizes of a purplish colour. The swelling in the limbs, as a rule, remains throughout the duration of the disease, which is usually of a week or a fortnight in extent, but it may be longer, the swelling then gradually disappearing. A sudden disappearance of the swelling, particularly if it is accompanied by colicky pain, is most unfavourable, and frequently results in death in a short time, due to intestinal complications.

The patient is often much depressed and debilitated. A certain amount of fever is usually present. The temperature may be high—105° F. It frequently drops when the swelling occurs, and at the same time the animal becomes brighter in appearance.

The appetite is, as a rule, fairly good. If it is entirely lost it is a most unfavourable sign.

Treatment.—Healthy hygienic surroundings, good nursing, and nourishing food, such as oatmeal gruel, linseed gruel, milk, if the patient will take it, are of the greatest importance.

The drug that has found the greatest favour with veterinarians in the treatment of this disease is oil of turpentine, given in 1-ounce doses three times a day, mixed with milk and eggs. A disadvantage in the employment of this drug is that it has to be administered as a drench, and drenching is not only difficult, but also dangerous, when the disease is affecting the animal's head. Tincture of perchloride of iron is often given in combination with the turpentine.

In cases where it is impossible to drench animals by reason of the swellings, Dickerhoff's treatment is adopted. This consists of the intratracheal injection of Lugol's solution of iodine in 1½ to 2½ ounce doses twice daily. The syringe employed should be first boiled and the skin on the neck disinfected thoroughly. The needle is inserted between any two of the cartilaginous rings of the trachea, and the iodine injected slowly. During the operation the horse's head must be held up to prevent the iodine gravitating towards and into the throat. If it is allowed to do so, it causes a violent fit of coughing.

A difference of opinion exists as to the efficacy of this treatment. In the hands of some practitioners excellent results have been obtained, it being only found necessary to continue the injections four or five days, or to put the patient on to a single daily injection in even less time than that.

WHITE SCOUR

It must be definitely understood that diarrhœa—or undue looseness of the bowels—may be a common result of some error in diet, or a result of simple indigestion, and at first may be nothing more than an attempt of nature to relieve the stomach and bowels of offensive and irritating contents.

But the form of diarrhœa dealt with here is known in Europe as " white scour," from the nature of the dung, and is caused by the presence of one or more micro-organisms or bacteria.

History.—The disease has been clinically recognised for a long time, and was put down to dietetic errors. At the end of the last century it was very prevalent, and its ravages were severely felt in Ireland, where it was investigated by Nocard in 1901. In 1891 Jensen began the scientific investigation of the disease in Denmark, and since that date it has been studied by a great many other investigators.

Nature.—All investigators agree that the disease is of bacterial origin, but a considerable difference of opinion has existed as to the actual causal organism and the manner by which the disease is contracted.

Nocard believed that the disease was caused by an organism of the fowl cholera type, and that infection took place at the time of birth by the umbilical vessels; but it appears established by Jensen and others that white scour is caused by intestinal bacteria resembling the *Bacilli coli communis.*

The disease has not been bacteriologically investigated in South Africa, but a disease in calves exists that answers to the description of white scour given by the before-mentioned investigators.

Nocard, in his investigations of the disease in Ireland, recognised two forms or phases of it—viz., (1) An acute septicæmic form, which appears shortly after birth. This form has been observed in South Africa, but is not so frequently seen as other forms. (2) A lung form of the disease, which occurs when the calf is about six weeks old. This form is frequently seen in this country.

According to Jensen, various forms may be distinguished in calf scours, according to their causes, and they may be divided into two groups, depending on whether they are associated with bacteriemia or are free from it, and under the heading of the forms due to bacteria he (Jensen) mentions paracoli bacillosis or pseudo-coli bacillosis. This disease attacks not only suckling calves, but also older calves and young cattle. It runs the course of an enteritis, frequently with hæmorrhagic tendencies.

My experience of the disease clinically agrees with that of Jensen, in that I have found the disease frequently in older calves and young animals, and that it is very common to have " bloody scours " in such animals, also even in fairly young calves, in place of the usual " white scour."

Pending bacteriological confirmation, I think it may be assumed that the different forms of scour in calves seen in South Africa are probably caused by as great a variety of organisms as those found in the disease in other countries.

White scour is an acute contagious, infectious disease. Calves are affected more frequently than other young animals, but the disease also occurs amongst foals, lambs, and pigs. The many bacteriological studies of the disease have led to the belief that in the majority of cases the disease is caused by the *Bacilli coli communis*, or by one of its several virulent varieties. The colon bacillus is almost a constant inhabitant of the intestinal canal of most animals and of man. Under normal conditions the bacteria multiply in the intestinal canal only to a moderate extent, and are harmless, as they are not capable of attacking the mucous membrane of the healthy intestines. If, however, the resistance of the tissues is weakened from some cause (cold, poor nutrition, etc.), the bacteria penetrate into the intestinal wall, and later also into the distant organs, as well as into the circulation, and produce in this manner an affection of the animal. Colon bacilli which have obtained a higher virulence in the animal body retain it for that respective species of animal. Leaving the animal body, they may again be taken up by the digestive tract of other new-born animals and produce the disease.

Natural Infection.—New-born animals are more susceptible or predisposed to the disease immediately after birth. The animals become affected in the majority of cases within twenty-four to forty-eight hours; much more rarely, from three to eight days after birth. The withholding of colostral milk from the calf, and the immediate feeding of boiled milk after birth, results frequently in the appearance of the disease. In infected

stables the virus may be present in the vagina of the mother, on the floor of the stable, or in the litter, from which the external genitals and the udder of the mother become contaminated. The infection results usually during the sucking of the contaminated udder, but also has taken place during parturition and later through contact with litter, from licking of the stable walls, etc. The affected animals may then infect their surroundings with their excrements and urine, thus increasing the possibilities for infection of animals born later. The contagion may retain its virulence in infected stables for a long time, which may explain that in some premises, when nothing is done for the prevention of the disease, it reappears from year to year.

Symptoms.—The new-born cease to suck, and stand in one place with a staring look. They are depressed, and lie down a great deal of the time. The diarrhœa appears. The expelled very fluid and fœtid fæces are yellowish in calves; later greyish-white, frequently foamy, and contain lumps of clotted milk, as well as blood-streaks (white and red diarrhœa). The parts around the anus are soiled, and the hair or wool is matted. The consistency is at first slightly mucous, later watery. The odour is intensely sweetish, later penetrating and putrid. The fæces are passed at first under severe straining; later it is painless, and towards the end of the disease involuntary. After the passages the animals stand in one place with feet drawn together, arched back, and lowered head, or they lie for a long time motionless on the ground.

The weakness increases during the course of the disease. The eyes sink into their sockets, the hair is rough, the skin is sticky from perspiration, the body disseminates a disagreeable, sweetish odour, and, finally, the greatly emaciated animals enter into a comatose state and die.

The carcases are greatly emaciated and anæmic; the mucous membrane of the stomach appears highly reddened; the small intestines contain a very fœtid yellowish or dirty-grey, and sometimes reddish, fluid contents which may be mixed with mucous flakes and a great quantity of gas. The intestinal mucous membrane is highly reddened, covered with sticky mucus, and may be studded with hæmorrhages.

In the lung form of the disease that occurs in calves about six weeks old the foregoing symptoms are not all present or so pronounced, and the question arises, which cannot well be decided clinically, whether such cases are white scour, or whether they are cases of contagious pneumonia of calves caused by an

organism of the fowl cholera type, or whether it is a case of the former disease being complicated by the latter.

Treatment—*Medicinal.*—Medicinal treatment of affected calves is mostly unsatisfactory. A common treatment is to commence by giving a dose of castor oil of from 1 to 4 ounces, according to the size of the animal, to clear the bowels, and following this with a mixture such as the following: Chlorodyne, 6 ounces; prepared chalk, 4 ounces; powdered ginger, 1 ounce; water to 24 ounces. Of this, one to four tablespoonfuls are given twice daily.

A very simple remedy for which good results have been claimed in cases of diarrhœa in calves is prepared from the pomegranate as follows: Twelve ripe pomegranates (unripe ones are said to act as well) are boiled in 1 gallon of water until the fruit sinks to the bottom. It is then allowed to cool. A breakfast cup of the liquid is the dose for a calf; a second dose is said to be rarely required.

Good results are likewise claimed from the administration of a dose composed of Epsom salts and bicarbonate of soda in equal quantities, the dose to be regulated according to the size of the calf, about an ounce of each being given to a calf a few weeks old. Sour milk has also been used with success.

Another treatment which has afforded relief in some cases is the administration of six or nine drops of creosote in sweet oil, milk, or gruel twice a day.

Tanniform, in doses of from 15 to 45 grains, made into a paste with honey or treacle to which $\frac{1}{4}$ grain of calomel has been added, is sometimes very satisfactory.

Whatever medicinal treatment is adopted, an attempt should be made to maintain the calf's strength by allowing it to remain with its mother and take nourishment as it requires it, or by the administration of milk, eggs, and mucilaginous gruels, such as linseed tea, oatmeal gruel, etc.

In the lung form of the disease good results are said to have followed the intravenous injection of tallianine, an ozonised turpentine prepared by Brigonney, Père La Plaine, Saint Denis, France.

Preventive.—In order to prevent the infection of the newborn, great cleanliness should be observed in the stable and about the cow. If the disease has been in existence in the premises before, the stable and floor in particular should be thoroughly disinfected every few weeks. A clean bed of hay or straw should be provided for the cow to calve upon. The cow's vagina should be washed with disinfectant before and after calving, and the udder should be washed before the calf is allowed to suckle.

Nocard, who believed the organisms entered the body by way of the umbilical vessels at the moment of birth, in addition to the foregoing precautions, recommended the ligaturing of the umbilical cord, similar to what is done in navel ill (see Navel Ill), and painting the stump with tincture of iodine.

The foregoing preventive measures to a greater or less extent have been practised at different times on infected premises in Rhodesia with, on the whole, favourable results. Great difficulty arises in being present when the cows calve, and ligaturing of the cord at the proper time. Painting the stump with fairly strong permanganate of potash solution has been found to answer as well as iodine.

In badly infected herds and premises the best preventive measure I have found is to allow the cows to calve out in the veld, but even then an odd calf will develop "white scour"; but it will reduce the cases to a minimum, particularly if the calves are allowed their liberty, and not congregated together in a calf pen. Another preventive measure is to keep the calves in healthy, thriving condition. If the animals become debilitated from any cause, such as ticks, improper or insufficient food, they are predisposed to the disease.

Inoculation.—Jensen prepared a serum through the horse by inoculation that, as a monovalent serum, proved only partially successful; but with a polyvalent serum he had better results, although these were not always uniform in practice. On some premises where, in previous years, not a calf had been reared, the serum reduced the deaths to nil; in other cases the deaths were reduced from 70 to 20 per cent. The indications are that to obtain the greatest success, serum which is autogenous polyvalent should be used in place of stock serums.

EPIZOOTIC LYMPHANGITIS

Synonyms.—African Glanders, Neapolitan Farcy, Japanese Glanders or Farcy (French, *Farcin de rivière*).

History.—The disease was formerly only observed in the southern part of Europe, along the shores of the Mediterranean; latterly it has been found to exist in a much wider circle in Europe, also in Asia and Africa. In 1902 it was transported with shipments from India to South Africa, and from the latter place to England by army horses returning at the conclusion of the Boer War. It gained wide prevalence in several garrisons, and was subsequently disseminated by cast army horses to a considerable extent throughout Great Britain.

No doubt, the movements of the Imperial Army horses carried the disease far and wide in South Africa; but the shipment of horses from India cannot be accused of having primarily introduced the disease into South Africa, as some years previous to 1902 Dr. Hutcheon, Chief Veterinary Surgeon, and Messrs. Borthwick and Henning, of the Colonial Veterinary Department, described the disease as existing at Koonap Heights, Cape Colony; and its existence had also been demonstrated in Rhodesia, in the Umtali district, where it was known under the name of pyæmia, and, for the reason of its prevalence, it was declared a contagious disease, and scheduled under the Animals' Diseases Ordinance in 1901, and horses, mules, and donkeys were prohibited movement from the infected area.

Nature.—Epizootic lymphangitis is a chronic contagious disease of horses, mules, and donkeys, and in exceptional instances cattle are also attacked. The agent responsible for the spread of the disease is a yeast fungus, *Cryptococcus farciminosus*, first described by Rivolta, an Italian investigator. This organism is found in the discharges from the characteristic sores which make their appearance during the course of the disease. The disease is characterised by purulent inflammation of the subcutaneous lymphatic vessels, and of the neighbouring lymph glands.

Symptoms.—Inflammation of the lymphatic vessels may take place on any part of the body, but it frequently begins on the limbs or the region of the scrotum and udder. In a number of cases it commences at the site of a wound, which at first takes on an unhealthy aspect; the surrounding tissues become swollen and hardened, and the sore exudes a watery pus. This condition is followed by swelling of the adjacent lymph vessels, which form prominent, painful, branching cords, along which tumours form, usually about the size of a Barcelona nut or larger. These, at first firm, gradually soften and rupture, discharging a thick, creamy pus. It is well to note the nature of the discharge, because the abscesses are very similar to what are known as "farcy buds," manifested in some cases of glanders, but in the latter the discharge is of a "sticky" nature. After breaking, the abscesses transform into ulcers with indolent, easily-bleeding, granulating borders. If these sores are neglected, large abscesses frequently form in the adjacent groups of lymphatic glands, which discharge, when opened, great quantities of pus. Sometimes abscesses form internally in the lungs or other organs. In such cases death inevitably follows.

In animals that have been treated and that have recovered from the disease, scars are left at the site of the abscesses, the pattern of these scars forming a reliable index that the animal has suffered from the disease.

Diagnosis.—The disease that epizootic lymphangitis is most liable to be confounded with is glanders, and no doubt a number of animals suffering from the disease have been diagnosed as suffering from the farcy form of glanders and been destroyed, which mistake, if destruction for epizootic lymphangitis is not provided for by law, is a mistake in the right direction. In addition to the difference in the sores and their discharges, animals suffering from epizootic lymphangitis are frequently in good physical condition, whereas in the farcy form of glanders the reverse is usually, though not invariably, the case. The demonstration of the *Cryptococcus farciminosus* by the microscope in the pus, and failure of infected animals to respond to the Mallein test, as well as the experimental inoculation of guinea-pigs, are the final scientific means of determination in cases of doubt between the diseases.

Methods of Infection.—Natural infection takes place in the great majority of cases from superficial wounds, harness abrasions of the skin, and infected harness transferred from one animal to another is a fruitful source of infection. Flies may also carry the

disease from a diseased to a healthy animal, and there seems to be little doubt that ticks are capable of communicating the disease. Mr. Henning first drew attention to the fact that the initial lesions of the disease frequently occurred at the sites where ticks become attached, and Mr. Jarvis, Government Veterinary Surgeon, when dealing with the disease at Umtali, came to the conclusion that this was so, and gave it as his opinion that the male of the tortoise-shell tick (*Amblyomma variegatum*) was a great offender. The eradication of the disease following the destruction of the tick by the regular systematic dipping and spraying of animals at Umtali apparently corroborated his theory.

Treatment.—The successful treatment of the disease depends to a great extent upon the site affected and upon treatment commencing at an early period. If it is possible to dissect out the cords with the tumours thereon and the adjacent lymph glands, success may be expected. Failing this, prompt incision of all abscesses, the evacuation of all pus, and dressing of these and all ulcers with an astringent antiseptic. Iodine dissolved in pure carbolic acid is good—so are powdered crystals of permanganate of potash—as a caustic, but care must be taken to prevent this getting on the skin of the animal; 2 or 3 drams of chloride of zinc in a pint of water also makes a good dressing, but the treatment may be, and usually is, a prolonged one, and animals that have apparently recovered frequently break out again. Owing to this fact, and to the risk of the disease being conveyed from the sick to the healthy animals, it is in most cases advisable and economic to both the owner and the community generally to destroy animals suffering from the disease.

Preventive measures are indicated by what has already been said, and should include thorough disinfection or destruction of all articles and places that the pus may have come in contact with.

ULCERATIVE CELLULITIS IN THE HORSE

Synonyms.—Ulcerative Lymphangitis, Pseudo - glanders, Pseudo-farcy, Suppurative Lymphangitis.

History.—This disease was first studied by Nocard in 1892. The name "ulcerative lymphangitis" that was first used is somewhat confusing with epizootic lymphangitis. As these are separate and distinct diseases, the name of ulcerative cellulitis that is used at present is more distinctive, and therefore preferable. This disease is included in the list of notifiable diseases in the Union, but up to the present has not been observed in South Africa. The disease occurs in France, and has been observed in the ox in Belgium. During the recent Great War it was one of the three or four diseases that caused the greatest amount of loss to the British Expeditionary Force in France.

Nature.—It is a disease of the horse characterised by an ulceration of the skin and the superficial lymphatic vessels, which in their course are the seat of abscesses caused by the Priesz-Nocard bacillus.

The Priesz-Nocard bacillus exists as a saprophyte in nature, and apparently gains entrance to the system through wounds and abrasions of the skin, particularly through those on the extremities. Cracked heels in winter, and sores caused by a horse getting its foot over the riem, provide ready means of entrance for the organism, but wounds or abrasions on any other part of the body may also become infected.

The Priesz-Nocard bacillus, which also causes caseous lymphadenitis in sheep, is compared with regard to its effects on the system through the toxin secreted to diphtheria, and the injection of antidiphtheric serum has been found to modify the properties of the toxin of the Priesz-Nocard bacillus.

In conjunction with the Priesz-Nocard bacillus in cases of this disease, other organisms are frequently found, particularly streptococci and staphylococci. In some cases these latter organisms predominate, and may be apparently the only ones present.

Symptoms.—The onset of the disease is frequently denoted by lameness—in some cases a very severe form of lameness—and the seat of the disease, very commonly between the hock and fetlock or the knee and fetlock, is swollen and simulates an ordinary sprain; it is also hot and painful. The first diagnostic symptom to appear is often a small swelling about the size of a pea, or larger. In hairy-legged animals this may be overlooked, and the first thing seen is a string of bloody pus down the limb, as a result of the swelling having suppurated and burst. As may be gathered from the foregoing, a suppuration of this nature develops very rapidly, which it does, twelve to eighteen hours being a sufficient length of time for it to do so. Within two or three days a sloughing of necrotic tissue occurs at the seat of the ulcer, and the disease tends to spread by the appearance of similar suppurating swellings in the vicinity of the original one. If they occur close together there is a tendency for them to run together and form a large open sore. In other cases these swellings follow the course of the lymphatics, and appear in chain formation, resembling epizootic lymphangitis and farcy. The ulcers show no tendency to heal quickly with ordinary antiseptic treatment, and, in fact, except by bacteriological examination of the pus or the Mallein test, great difficulty is experienced in some cases in clinically diagnosing between these diseases.

Treatment.—Treatment of the disease during the Great War was found on the whole to be mostly unsatisfactory owing to its nature of recurrence at short distances from the original seat, and the difficulty of healing the sores. Ulcers that apparently dry up and heal often break out again, either within a few days, or it may be weeks.

Treatment consists in clipping the hair from the affected part, and thoroughly cleansing the part with an antiseptic, painting the wound with tincture of iodine, packing it with gauze soaked in antiseptic, and covering with a bandage. The dressing to be removed and freshly applied twice daily, and, of course, all dead tissue must be removed from the wound. Ordinary solution of cupri sulphas seems to have most beneficial results.

Latterly, a vaccine treatment has been evolved, the material employed being a vaccine prepared from the Priesz-Nocard bacillus. This treatment gives promise of perhaps ultimate success, but at present it is only in its infancy.

In the British Expeditionary Force in France during the later stages of the war it was found unprofitable to treat animals which were badly affected with this disease, and all animals showing

the undermentioned lesions were destroyed on arrival at the Base Hospital, and no attempt at treatment carried out:

A. Any animal showing ulcers above the knee or hock.

B. Any animal showing more than two small ulcers below the knee or hock.

C. Any animal showing a large or extended ulcer below the hock or knee.

Even by the use of polyvalent or autogenous vaccines, and careful local treatment in very mild cases of the disease, a permanent cure could never be assured.

SWINE ERYSIPELAS

Synonyms.—Red Soldier, Blue Disease, The Purples, Diamonds.

History.—Swine erysipelas has probably existed for many centuries, but was, until comparatively recent years, mixed up with, and mistaken for, a form of swine fever. It is only within the last twenty-five or thirty years that the term has become common in the English language. It was taken from the German name of "Schweine-Rothlauf," and it was in Germany that the disease was first identified as a separate one of the pig. It was differentiated on clinical grounds before its bacterium was discovered by Löffler, in the early eighties. Löffler's investigations firmly established the fact of the disease being separate and independent.

It is prevalent in most countries on the continent of Europe in epizootic form, and in Germany, Austria, Denmark, and France many thousands of pigs succumb to its ravages annually. In Great Britain sporadic outbreaks occur in which a great number of pigs may die. The disease is constantly present there in its chronic form. It is said not to exist in the United States of America. In South Africa, up to the present, the existence of this disease has not been demonstrated.

Nature.—It was, until a few years back, regarded entirely as a contagious disease; but, as a matter of fact, it is in the great majority of cases of sporadic occurrence. The bacillus is a common soil saprophyte. It is said it has been found in the mouths and tonsils of healthy pigs. In Great Britain the disease is, as a rule, sporadic, and often exhibits no tendency to spread by contagion or infection; but large numbers of pigs are sometimes infected simultaneously or in quick succession, and it is possible that these cases are spread from pig to pig. Of course, on the other hand, they may be simultaneously infected or in quick succession sporadically. In explanation of the sporadic outbreaks, it has also been suggested that there may be strains of the swine erysipelas bacterium in the soil or in the outer world that are non-pathogenic, but, owing to some unknown cause, these

strains become virulent, and perhaps a single pig becomes infected, and the disease, thus started, then becomes contagious

In temperate climates swine erysipelas has a markedly seasonal occurrence; comparatively few cases of acute or subacute forms occur in the winter. The disease apparently crops up in the summer and disappears in the autumn. This is probably due to the higher temperature being favourable to the propagation of the bacteria in the soil and in the manure of the sty. In acute cases the disease is in a measure septicæmic, and the bacilli are sufficiently plentiful that they can be detected in the blood by microscopical examination; but they can be detected still more certainly by culture. As a rule, the number in the blood is only moderate, and never approaches the quantity of organisms present in a case of anthrax.

In subacute and chronic cases the bacilli cannot be found in the blood by microscopical examination, and in quite chronic cases the blood may not be infective when experimentally inoculated. The bacilli are in the lesions, particularly those of the heart.

It is essentially a disease of the pig, but man is susceptible to infection by inoculation, and many cases have been recorded. It is usually contracted through a wound at a post-mortem examination, and, as a rule, its effects are not very serious, being generally confined to an inflammatory lesion of the hand or arm, but it has caused death.

So far as is known, the pig is the only domestic animal to contract the disease naturally. The mouse and the pigeon are highly susceptible by inoculation, and death occurs in from three to five days, the disease being always septicæmic, though the bacilli are not enormously abundant in the blood. Subcutaneous inoculation of the rabbit causes only a local inflammatory lesion; death rarely occurs. The guinea-pig is not readily infected, but the virulence for this animal can be greatly intensified by passage from one guinea-pig to another.

Probably the natural method of infection in the pig is by ingestion, but experimental feeding results are remarkably uncertain.

A remarkable point in connection with the disease is that young pigs under three months old, and adult pigs, are comparatively insusceptible. The disease is principally seen in pigs from four to eight months old, and at times, even where numerous cases are occurring, the little pigs and the sows may entirely escape. The susceptibility varies with the breed of pigs. Pure-bred English pigs, when imported into the continent of Europe, are especially susceptible.

Symptoms—*Acute Form.*—This is the form of the disease that prevails on the continent of Europe. It is septicæmic in its nature, and the mortality is from 50 to 80 per cent. The visible illness does not exceed a day or two; the attack is sudden and rapidly fatal. The symptoms are not very characteristic, being the usual signs of acute illness in the pig. The animal refuses food, lies buried in the straw, and the temperature is high. The skin becomes discoloured; bright red spots, somewhat raised, appear on the throat and the under-surface of the body; or the discoloration may be diffuse, covering the greater portion of the body, which becomes a dark rose or purple-brown.

The post-mortem lesions are inconsiderable. Inflammation of the stomach and intestines may be present, but there is no necrosis or diphtheritic deposit. The spleen is enlarged. The spleen pulp is a favourite habitat of the bacillus.

Subacute Form.—In this form of the disease, in addition to a moderate degree of fever and loss of appetite, a conspicuous skin eruption occurs which has often been mistaken for urticaria (nettlerash) in young pigs. In older pigs (six months old) the skin eruption takes the form of distinctly raised, circumscribed patches of the size of a penny-piece. They may be rounded, but are usually diamond-shaped. The common sites for these lesions are the neck, back, shoulders, or buttocks. They are livid in colour, and may remain distinct, but usually some of them tend to run together, forming large, irregular, raised patches. The hock and knee-joints are often swollen, and walking, if the animal is compelled to do so, causes it great pain.

Usually, about the fourth day the discoloration of the skin disappears, and the animal quickly recovers. On the other hand, sometimes an actual vesiculation of the skin lesions occurs, and in severe cases there may be extensive necrosis of skin; and, if the pig survives, sometimes the ears are shed, or large patches of dried skin are exfoliated.

Chronic Form.—In this form of the disease the history of the case is frequently that the animal survived an acute or subacute attack, but remained in an unthrifty condition; but in a great many cases the pig is found dead, and has not previously been observed to be ill.

In other cases the animal will be noticed to be ill. The breathing will be accelerated; a soft cough may be present, accompanied by a certain amount of skin discoloration, particularly of the ears and groin; the appetite is capricious, and the animal constipated.

As the disease progresses, breathing becomes more difficult, and the skin discoloration becomes intensified, the animal dying within a week or fortnight.

The post-mortem appearances are, usually the carcase is more or less wasted with skin discoloration at the before-mentioned sites. Fluid may be found present in the chest cavity, and the lungs in an œdematous condition. The lesions of the disease are in the heart valves. These are usually cauliflower-like in appearance, and are more frequently found on the mitral valve, but are often found on the aortic also.

Treatment.—Treatment is of a preventive nature. Affected animals should be isolated and disinfection of sties, etc., carried out.

Various methods of preventive inoculation have been tried. The favoured one, and the most successful one, is very similar to that known as the simultaneous inoculation for rinderpest, the agents used being blood-serum obtained from a hyper-immunised pig, and a pure culture of the causal organism.

The procedure is as follows: When the disease has broken out in a herd of swine, the animals receive at once an injection of from 10 to 20 c.c. of serum Ten days later the animals are injected with 5 to 10 c.c., according to size of serum, and 0·8 c.c. of pure culture. Twelve days later the animals receive a further 0·8 c.c. of pure culture without any serum.

A variation of this method is to inject serum in doses of 1 c.c. to each 20 pounds body live-weight, and follow this in six or seven days by an injection of 0·5 c.c. of pure culture. This method has been practised largely, and has been found entirely satisfactory.

TETANUS

Synonyms.—Lock-jaw (French, *Tétanos*).

Definition.—Tetanus is an acute infectious disease resulting from an intoxication of the nervous system, characterised by persisting, spasmodic contractions of all the muscles of the body, or of groups of them.

History.—The theories assigning the cause of the disease were many until comparatively recent years. In 1884 Necolaier obtained positive results by inoculation of garden earth into rabbits, guinea-pigs, and mice, and at the same time recognised the bacillus causing the disease. In 1887 Nocard established the identity of the cause of tetanus in horses. In 1889 Kitasato grew the bacillus in pure culture.

Nature.—Tetanus, or lock-jaw, is a disease of comparatively rare occurrence amongst animals. In South Africa, during twenty years' experience, I do not suppose I have seen more than ten cases.

The disease is caused by an organism called the *Bacillus tetani*, which commonly exists in the soil as a vegetable feeder, and is frequently present in the bowel contents of herbivorous animals without doing any harm to its host. This organism has a very wide and common, but unequal, distribution in soil. It is abundant in cultivated and manured soils, and is more abundant in some places than in others, judging from the frequency of cases occurring in certain places. The dust of streets sometimes appears to be specially dangerous. This may be due to the horse fæces that is mixed with it, and animals engaged in sanitary services are especially liable to contract the disease. So far as is known, all the mammals are more or less susceptible to the disease, but horses furnish the largest proportion of cases. This is probably due to the horse being more susceptible to wounds. Next to the horse comes mules and donkeys. The disease is more common in sheep than in the ox. It is rare in the pig, and more so in the dog.

Numerous experiments go to prove that normally the disease cannot be conveyed by ingestion or inhalation. When animals

are fed on large quantities of the organism or its toxin, apparently neither are absorbed or destroyed; they just pass through the alimentary canal. The great majority of natural cases of tetanus in animals and man are the result of wound infection—the so-called traumatic tetanus—but in both veterinary and human practice there are cases in which no wound is discoverable. In veterinary practice such cases are called idiopathic tetanus, and in human practice rheumatic tetanus. Possibly in the horse the disease may be due to the wound being a very small one, and that has healed by the time the symptoms of tetanus set in; but there is reason to believe the tetanus spores are transported from wounds by the general circulation to various parts of the body, and persist there for a length of time without multiplying, and it is believed such latent cases may develop tetanus. When the fracture of a bone occurs, the animals may suddenly develop tetanus. It is also believed the spores in such cases may start to germinate and multiply without any apparent cause.

In natural cases the period of incubation in the horse appears to be about eight days, but it varies, and in exceptional cases may occur in two days.

Symptoms.—The symptom that gives the name of lock-jaw to the disease is due to the fact that the muscles that close the jaws are very much stronger than those that open them, so that when a tug-of-war of muscular contraction occurs, the former win easily, resulting in the mouth being firmly closed, and unable to be opened even by applying considerable force. This condition of trismus, as it is called, is not always present; in some mild cases a greater or less movement of the jaws can take place. The symptoms presented to a great extent depend upon the set or sets of muscles that are principally affected, but the following are usually present: The animal presents a stiff, wooden appearance, with the head poked out, the tail elevated and quivering, the muscles hardened, and animal is stiff if made to move; great nervousness and excitability; and perhaps the most diagnostic of all symptoms is that when the head is slightly elevated by placing the hand on the chin and raising it, the haw is drawn across and may entirely cover the eyeball. The nervous excitability is most marked in the great majority of cases, the animal keeping its ears on the alert all the time, and a sudden noise, such as a hooter, causes it a great shock. The haw shoots across the eye, the muscle stiffens in a spasm, and the animal may fall down with the fright, and if it does the case is usually a hopeless one.

Treatment.—In places where tetanus is of frequent occurrence, and where the worth of the animals justify the expenditure, it is the practice to administer antitetanic serum to animals before and during the period of recovering from operations or wounds. This serum is purely of preventive value. It has no curative effect once the symptoms of the disease have appeared, but it is possible to get a serum so strong that 10 c.c. of it will make a horse for the time being unkillable by experimental tetanus, so as a preventive it is of great value. Certain American practitioners have claimed satisfactory curative results from the use of serum in large doses (4 to 6 ounces daily), but, needless to say, this is a very expensive treatment.

The treatment of animals showing symptoms of this disease consists in keeping them as quiet and undisturbed as possible. An isolated, darkened loose-box is the ideal place, and if it is impossible to prevent the animal getting shocks from noises, it is advisable to plug its ears with cotton-wool to prevent it hearing them. If it is able to take any nourishment, sloppy food should be given with a view to keeping its bowels in a normal condition, and Epsom salts and saltpetre may be put in its food or water, but if this puts it off its food it should be discontinued. In no case should one attempt to drench an animal suffering from tetanus, since the excitement does much more harm than any medicine will do good. Electuaries of belladonna is a favourite remedy with some; others employ nerve sedatives—morphia, chloral, bromide of potassium. I have tried most of these things, and have come to the conclusion that an equally good, if not better, treatment is for the owner to put the key of the loose-box door in his pocket, and only go there once or twice a day himself for the purpose of giving his patient the necessary food or water, and trust to nature to bring about recovery.

Recently a new treatment for tetanus has been employed with very good results, according to the reports. It is certainly very simple and inexpensive, and consists of subcutaneous injections of saturated solutions of Epsom salts; 40 c.c. of such a solution are injected (20 c.c. behind each shoulder) daily for about five days, and afterwards 20 c.c. are used daily for a few days.

QUARTER EVIL

Synonyms.—Black-quarter, Black-leg, Quarter Ill, Strike, etc. In South Africa it is frequently called Sponge Sickness (Dutch, *Sponsziekte*).

History.—The disease has been known since the dawn of veterinary science, and in the early days was thought to be a form of anthrax. Charbert, in 1790, appears to have been the first to clinically distinguish the disease from anthrax. A century later the causal organism was demonstrated, and a method of vaccination was devised, since when much work has been done in connection with the disease by numerous investigators. The disease has been known in South Africa from the early days, but its history and nature in Southern Rhodesia, owing to it being apparently, to all intents and purposes, a disease new to the country, are worth recording.

Matabeleland, that portion of Rhodesia in which the disease was first observed, had been occupied by the British for about seventeen years before the disease was discovered in July, 1911. During those years there had always been a few qualified veterinary surgeons in the country, but owing to rinderpest sweeping through the territory in 1896, later followed by lung-sickness (pleuro-pneumonia) and East Coast fever, there had not been a great number of cattle in the country. In July, 1911, two outbreaks of the disease occurred—one at Redbank, to the west of Bulawayo, and the other at Insiza, to the east. Less than twenty head in all died at both places. The treatment adopted was simply to move the cattle to another portion of the farms, and the disease stopped. Nothing further then occurred until December, 1916, when the disease was diagnosed in the Plumtree district. Reports had previously been received that the disease was prevalent, and was killing a number of cattle in the Tati Concessions. As in 1911, the practice of moving the cattle was again adopted. It answered in many cases, but the disease killed a great number of cattle, and preventive vaccination was introduced. Naturally, more attention was directed to the history and nature of the disease in 1916, and enquiries were made through

the native department as to the natives' knowledge. The result was, as is frequently the case where natives are concerned, somewhat conflicting, but finally the weight of opinion was to the effect that the natives did not know the disease; but it was also fairly certain that odd cases had occurred at different times, at least, during the European occupation of the country, and these had been attributed by Europeans and natives alike to be due to snake-bite. No evidence was forthcoming to show that the disease had even existed in any herd in the country destroying similar numbers to what it did in 1916.

In 1917 deaths reported totalled 1,335; probably a number unreported also occurred. In 1918 there were 1,104 deaths, but, in view of the fact that quarter evil is always regarded as a sporadic and a non-contagious disease—probably quite correctly so—it is interesting to note its progress through the country. Although it did not exactly spread from farm to farm, it entered Southern Rhodesia on its south-west border in 1916. It did not stop summer or winter, and now, at the end of 1919, it has reached the north-eastern boundary of the territory. In view of its steady spread—every new outbreak farther north or north-east, and many incidents that can only be accounted for in either one or two ways, and they are coincidences or contagion—one is moved to wonder whether the disease is invariably sporadic in character, or whether it is possible for the organism to assume another character.

In the majority of instances the disease attacks the young stock. It is expected to attack the fattest and best of the young stock, but in other cases quite thin stock die of it. In many outbreaks in Rhodesia aged animals—cows up to fifteen years old—and calves as young as six weeks old have died from it; but, as a rule, aged animals only die when the disease first appears in a herd, and such deaths may perhaps be explained away by the susceptibility of the individual.

The farms that were infected in the 1911 outbreak have not been any different to other farms in the present outbreak; in fact, upon the whole, they have been particularly free of the disease. Some have escaped without a case occurring. Much of the foregoing is so different to what one has been taught and one's experience of the disease in other lands, that it should prove interesting to watch the future developments of quarter evil in Southern Rhodesia.

Nature.—The disease is caused by an organism known as the black-quarter bacillus, or the *Bacillus chauveii*, which is a faculta-

tive parasite, and its normal method of existence is as a soil bacterium.

The disease occurs in all the great continents, but it has everywhere a regional distribution, and there exist large areas where the disease is unknown. It may also be present on one farm and not on another adjoining, and some fields on an infected farm are worse than others. Within quite modern times the disease has almost entirely disappeared from certain parts of England. Moorland and permanent pastures are the worst infected places, cropping and draining of the land being apparently unfavourable to the organism. The majority of the cases are sporadic, but the bacilli from a carcase may infect other animals; but such cases in Europe are believed to be very rare. Rhodesian experience leads one to suspect that they may not be so rare in South Africa.

The animals most susceptible to the disease are young cattle of from six to twenty-four months old; calves and animals over three years old rarely become infected in countries where the disease is enzootic or of annual occurrence. Sheep are also susceptible, and are affected at all ages. Like many other diseases, the individual susceptibility varies considerably, some animals apparently possessing considerable power of resistance to the disease.

The causal parasite is to a great extent confined to the tissues at the site of the disease, and is not, as a rule, in the blood-stream, although at times it may be found there.

The natural method of infection has been much disputed, but probably the weight of opinion is in favour of the theory that this takes place by ingestion, and that some unknown circumstances are present to render the ingested spore infective. One of the conditions favourable to the development of quarter evil that has always been recognised obtains at the time when the grazing is conducive to the young animal rapidly improving in condition or making blood, as it is sometimes called, and animals that are thriving the best in a troop are usually the first to succumb to the disease.

Symptoms.—The disease is rapid in its onslaught, often the first intimation of it being the finding of an animal dead. When a case is seen earlier than this, besides the ordinary symptoms of illness, with a dejected appearance and trembling, the animal, if made to move, will exhibit lameness or stiffness, and a swelling will be found, the hind quarters and the shoulder being the favourite sites for swelling, though it may occur on other portions of the body. This swelling at first is hot, painful, and

doughy, but it quickly becomes emphysematous—that is, blown up with gas. It crackles if you rub it, and sounds hollow if you tap it with your fingers. The skin over the swelling becomes painless, cold, and leathery—in fact, dead.

Post-mortem Appearances.—After death, if one cuts into this swelling, one will find emphysema present, the centre of the swelling more or less dry, and a sour, rancid odour—that is, if decomposition has not set in—and the flesh will be black in colour.

The only two diseases that are likely to be confounded with quarter evil are anthrax and snake-bites. In anthrax the blood is black and tarry-looking, and does not clot, and the spleen is always enlarged. In quarter evil the blood appears unaltered and clots normally, and the spleen is never enlarged. In snake-bites there is always the hole caused by the snake's fangs, the swelling is not so large, it is not emphysematous, it has not got a rancid, sour odour, it is not dry in the centre, and the blood clots quickly and firmly.

Treatment.—Quarter evil is one of the most fatal diseases, and when once an animal is visibly affected very few recoveries take place—at least, not more than 2 or 3 per cent.—so that there is no opportunity of judging from natural cases to what extent recovery from an attack confers immunity; but it is possible to confer a high degree of immunity in young cattle by several methods of protective inoculation.

There is no medicinal curative treatment known for the disease, and perhaps the matter of first importance to consider is prevention of the contamination of the soil by a proper disposal of the carcase of an animal dead of the disease. The carcase should be either buried or cremated. If burial is decided upon, a hole should be dug as near the spot the animal died upon as possible, and at least 6 feet deep, into which the carcase should be put intact, with any material or soil contaminated by discharge from the body, and the lot covered with quicklime if at hand. But if it is possible, a much better method is cremation (see Disposal of Carcases), not only for the reason that if thoroughly done it does away with all risk of future infection at the spot where the beast died, but also it does away with the possibility of the meat being removed by natives with the object of consuming it. Whether meat removed from one place to another in this way is capable of transporting the disease may be a moot point, but so many cases have occurred in Rhodesia of the disease breaking out on a farm when meat has been removed to that farm by natives from the carcase of an animal or animals that have died within

a few miles of it, that the stockowner will not believe that all such cases are simply coincidences, and is firmly convinced that meat is capable of transporting the disease.

Amongst the methods that have been adopted for preventing the disease, passing mention may be made of the one of getting on horseback and chivvying the stock across the country until they are tired out, the object being to take the condition off them, and the old method of setoning with cord, garlic, etc. These methods have not been employed to a great extent.

The favourite ones have been: (1) To move the animals to fresh pasture. (2) To vaccinate. (3) To do nothing. Good results have been claimed for all three methods.

To move animals to fresh pasture and kraals is a sound proceeding, but is attended with a certain amount of uncertainty in a colony where, in the majority of cases, the land is not even fenced off into large paddocks, and where no reliable opinion can be formed as to the locality where the animals contracted the disease. And cases have occurred where animals have remained perfectly healthy when grazing on summer pasturage of the farm, but when they have been removed to that portion of the farm that has been reserved for winter grazing, the disease has broken out amongst them. But, on the whole, the practice of moving the stock to new surroundings without doing anything else has been attended with fair results so far as the immediate stopping of the disease has been concerned.

To do nothing when the disease breaks out in one's stock may suggest laziness, but this may be a wrong conclusion to arrive at. Men who have had previous experience of the disease, although at the time they may not have recognised it, but regarded it as snake-bite, remembering that they only lost two or three head, are liable to think that the mortality will repeat itself and remain at the same number, which it has done in some instances; in others it has not, and preventive inoculation has had to be resorted to. It should be remembered that such stockowners may be confronted with the difficulty that they cannot ascertain the portion of their holding upon which the disease has been contracted, and that by the methods of inoculation practised they may by the urine and fæces of the inoculated animals further increase the infection of their land, a contingency to my mind not at all impossible.

Preventive Inoculation.—In South Africa Arloing's and Kitt's methods are the ones most frequently employed. Other methods are employed to a lesser extent. In Rhodesia a method

that has found considerable favour is known as filtrate inoculation.

Arloing's Method.—In this method two vaccines of different strengths are employed, which are prepared as follows from quarter evil muscle lesion containing only black-quarter bacilli.

A natural case is never serviceable on account of the accidental bacteria that are almost sure to be present; therefore, a large lesion is provoked in a sheep or in a young bovine animal by the inoculation of pure virus, and the animal is killed just when it is at the point of death, or a post-mortem is made immediately after death. The muscular tissue lesion is then cut out with sterile instruments. The dry and emphysematous portions, being the best, are selected and cut up into pieces the size of one or two fingers. These are then dried in a current of air or in front of a fire at a temperature of about 30° C. The muscle becomes dry and crisp, and can be preserved for years in this condition.

The dry muscle is next powdered by passing it first through a mincing machine, and then through a coffee mill. Next sterile water is added to it in sufficient quantity to convert it into a uniformly thick paste. All the muscle must be wetted.

First Vaccine.—To prepare this, the muscle paste is placed for seven hours in an incubator at a temperature of 100° C., spread out in a thin layer on a plate, and becomes a perfectly dry, brittle crust.

Second Vaccine.—This is the stronger, and is prepared in exactly the same way, except that the temperature is only carried to 90° C.

To prepare these vaccines ready for use, they are again passed through the coffee mill, then powdered still finer with a pestle and mortar, weighed out, and placed in the glass tubes, in which state they reach the stockowner with the printed directions as to use.

Kitt's Method.—This method differs from that of Arloing only in that the muscle powder is heated for six hours in a steam steriliser at 97° to 100° C. This method has a greater destructive effect on the spores than the dry heat has.

Only one injection is necessary under this method, which is commonly known in South Africa as the single vaccine, and Arloing's as double vaccine.

Filtrate Inoculation.—Professor Naoshi Nitta, of the Laboratory of Pathology and Bacteriology of the Tokio Imperial University, Japan, was the first investigator to perfect this method of vaccination.

In the July, 1918, issue of the *Journal of the American*

Veterinary Medical Association, Professor Nitta published the results of the vaccinations with black-leg filtrate in Japan and Korea in an article entitled "Investigations on Black-leg Immunisation," in which he concludes as follows:

"The filtrate of a pure culture of the black-leg organism confers a solid immunity on animals treated, and it has been already successfully used in thousands of cattle in infected districts. It is inexpensive, the material for preparation being aerobic cultures of the organisms in meat-piece broth, and its injection is not accompanied by the least danger, because the filtrate is quite germ free."

In comparison with many of the laboratory preparations and vaccines, the preparation of "filtrate" is comparatively simple. It is prepared by cultivating the black-leg germ in a special medium for a period until the maximum growth is obtained, and until the liquid becomes thoroughly charged with the product of the germs and of the meat contained in the medium upon which the black-leg germ acted. The product is then preserved and filtered through porcelain, which retains all the germs.

The successes attained in Japan and Korea by this method of vaccination have also been attained in the United States of America, and it was from the latter country that Rhodesia got its first information of the method and its first supply of vaccine.

The advantages that are claimed for it over the two first mentioned methods are that it does not contain the quarter evil germ in any form, and therefore cannot induce the disease under any circumstances. Further, it is claimed that the method prevents the reinfection of the soil through the discharges of sick animals and dead carcases, and that its periodical use tends to "wear out" the infection from the soil. But it is difficult to reconcile this latter assumption with the ability of the organism to live and thrive in soil independently of animals.

The immunity conferred by one inoculation is held to protect the animal for a year. Being a liquid, its application is certainly easier than that of the powder vaccines, which tend to clog the needle of the hypodermic syringe.

Other methods of preventive inoculation include, amongst others, aggressins, liquid cultures, and serum.

ANTHRAX

Synonyms.—Splenic Fever, Splenic Apoplexy, Malignant Pustule, Woolsorters' Disease (French, *Charbon ;* Dutch, *Meltziekte*).

History.—The disease was known by its present name (which means coal, on account of the black colour of the blood) from very early times, and is thought to have been the sixth plague of Egypt. Its contagiousness was first discovered in 1836 by Eilert; the causal organism was discovered by Pollender in 1855. These were artificially cultivated by R. Koch in 1876, and by Pasteur in 1877, whose investigations opened a new path for the theory of the infectious diseases in general.

The disease is world-wide in occurrence, and has been recognised from the earliest times in South Africa, and has caused heavy losses of stock in the provinces of the Union, particularly during the last few years, when it has apparently been on the increase. In contrast to this state of affairs, anthrax is one of the live-stock scourges that Rhodesia has suffered but little from. In 1898 some few cases occurred amongst a herd of imported cattle quarantined near the Ramaquabane River. The next outbreaks were in 1912: one on a farm a few miles from Bulawayo, where 6 cattle died; and another near Salisbury, in which 14 pigs and 1 donkey died. No further cases then occurred until 1917, when at three outbreaks near Salisbury, in the Mazoe district, in herds totalling 624 head 9 cattle died. In January of 1918 an outbreak occurred in the Makoni district, and 30 cattle died, the largest mortality that has been recorded in Rhodesia.

This fact of the absence of anthrax in Rhodesia is interesting, and causes speculation as to why it should be so. The optimists say the spores (*Bacillus anthracis*) that cause the disease are not present in the soil of the country, and every precaution should be taken to prevent their introduction. The pessimists hold that these spores are present, and one day, given favourable conditions, and when the cattle have greatly increased in numbers, more outbreaks of the disease will probably occur. There is something

in both of these theories. It seems difficult to believe that the disease has not been in the country when it is considered that it exists on practically all sides of Rhodesia, probably has existed for hundreds of years, and remembering the raiding and looting propensities of the Matabele and the other ways the disease could have been introduced. Referring to the two outbreaks—that at Ramaquabane, the other near Bulawayo—and perhaps also the outbreaks in Mashonaland, which may be held to prove that the disease is endemic, and considering the time that has elapsed since the two first-mentioned occurred, that stock have been running on these places since that time, and that no recrudescence of the disease has taken place, it may be maintained that Rhodesia is not a country very favourable for the existence of the anthrax spores; but this will remain to be seen.

Nature.—Anthrax is a specific infective bacillary disease caused by the entrance into the body of the *Bacillus anthracis*. It attacks all animals and human beings, and usually manifests itself in the latter in one of two forms. One is a malignant pustule on the hands, arms, or face. People who work with carcases usually contract this form of the disease. I have seen but one case of it, which was in a slaughterman, and it was not at all pleasant to look at. The other form is known as "woolsorters' disease," affecting the lungs, and, as the name implies, is contracted by inhaling the organism with other dust during the handling of the wool from anthrax carcases. There is also another form of the disease contracted by people eating the flesh of anthrax carcases. This is almost an impossible happening now in civilised countries, but here with our native population it is a different matter. A few years back, when the disease was prevalent in the Barotse Valley, in Northern Rhodesia, I was informed that many natives died from so doing. Apparently they contracted the disease somewhat similarly to pigs and dogs. It manifested itself in the throat and neck, and a hard carbuncular swelling appeared. A missionary in the locality, who, as frequently happens, was regarded by the natives as a medicine man, was consulted. On referring to his literature, he found that firing with a red-hot iron was the treatment recommended. Fortunately, his wife was an exponent of what is known as poker-work—that is, burning artistic designs on wood, such as the backs of chairs—and possessed an iron that was heated by other means than putting in a fire; so the husband got hold of this and started by burning lines on these swellings, and his first operations were

attended with success, and report has it he carried on an extensive practice.

In considering the nature of the disease, it is most important to decide to what class of organism the *Bacillus anthracis* belongs. In cold and temperate climates experience points to it being an obligatory parasite. If it multiplies outside the animal body, it is only to a limited extent. Cases of sporadic anthrax are extremely rare, if they ever occur. Outbreaks usually occur on farms from antecedent cases, and a long interval may have intervened (this point of the organism being able to maintain its existence outside the animal body, but not to multiply, is important). The insufficiency of temperature is the greatest obstacle to saprophytic organisms.

No multiplication of *Bacillus anthracis* occurs at a temperature below 50° F., and at 60° F. growth is very slow and meagre. Supposing multiplication was occurring on manure or soil, the *Bacillus anthracis* would soon be crowded out by other saprophytes that multiply at a lower temperature; but in India and other tropical countries, probably including portions of South Africa, in a hot moist atmosphere multiplication of the organism at the surface of the soil probably takes place.

Symptoms.—In cattle the symptoms of the disease may perhaps be described best in half a dozen words: sudden death and rapid decomposition of carcase. Usually a beast infected only lives a few hours. I have seen an animal feeding, suddenly stop, give a bellow, and drop down dead. Of course, some animals live longer; and whilst there are not, so far as I am aware, any absolute diagnostic symptoms in the living animal, the poor brute has an anxious and distressed look, as if it recognised the seriousness of its plight.

The symptoms and appearances of the disease in sheep are similar to those in cattle. In the horse an ante-mortem symptom is sometimes present in the form of swellings on the lower parts of the chest, abdomen, sheath, and around the anus, and occasionally of the throat and head, which increases in size in the course of a few hours. The pig and the dog also show a similar swelling of the throat and head, and the post-mortem appearances in these three animals to a great extent resemble those in the ox.

Post-mortem Appearances.—The post-mortem appearances are pretty well marked. The first thing noticeable is that there is—I may say, almost without exception—blood of a dark colour oozing out of the nostrils and from the anus mixed with black-coloured dung. Another most noticeable thing is the rapidity

with which the carcase decomposes. Very shortly after death the body is swollen up to almost bursting-point, due to putrefactive gases that have been evolved and escaped into the connective tissues under the skin, giving the carcase a drum-like appearance. Unfortunately there are other diseases in which death is sudden and decomposition rapid, such as quarter evil and lighting, and under our system of farming and temperature a beast that has been dead some time, when found, might present a similar appearance; therefore it would be necessary to go further with the examination. It is wrong in every way to knowingly open an anthrax carcase. In fact, in the Union it is forbidden to knowingly dissect the carcase of an animal dead of anthrax. Besides the great risk to the person opening the carcase, there is the danger of spreading infection to the soil and surroundings. But supposing, for the purpose of diagnosis, it is decided to open such a carcase, every precaution should be taken to prevent infection to the operator by seeing that he has no open sores or cuts, and by a liberal use of disinfectant. On laying back the skin, the veins will be found very full of dark and imperfectly clotted blood. It will probably be quite liquid, and tarry in consistence. The existence of firm clots of blood in the veins almost amounts to reliable evidence that the disease is not anthrax. On opening the abdomen, the enlargement of the spleen is the first thing to attract attention. This is almost without exception greatly enlarged, and when cut into the spleen pulp and blood run out and resemble tar more than anything else; but in South Africa the enlargement of the spleen as a diagnostic sign has not the same value as in many other countries, for the redwater type of disease also gives rise to enlargement of the spleen. In the latter type of disease, however, the spleen is not so soft, nor is it so tar-like as in the case of anthrax.

Diagnosis.—Probably the best way for a stockowner to obtain a reliable diagnosis of anthrax is to take a blood-smear from the dead animal and forward it for microscopical examination. But to be successful certain precautions have to be observed. Invasion of the blood by the *Bacillus anthracis* occupies only a very short interval of time before death. It often occurs that an animal may have a temperature of 107° or 108° F., and yet no bacilli can be found in the blood. This is an important point to remember: smears should be preferably taken from animals as soon after death as possible. Another point to remember is the class of animal dealt with. In the ox, sheep, mouse, guinea-pig, and rabbit the parasites are invariably present throughout the blood-

stream; in the dog and cat they are generally so; in the horse they may or may not be; in the pig they are not present in the general mass of blood at the time of death. There are exceptions to this rule, but they are found in the lesions and in the swollen throat, usually in great numbers in the lymphatic glands of the throat, also in the mesenteric glands; therefore in the pig, dog, cat, and horse it is advisable to take the smear from these places.

In taking the blood-smear from the dead carcase of a sheep or ox an extremity should be selected—the foot or the ear. A small slit in a vein in the latter organ that only allows one or two drops of blood to escape is the best manner. The reason—or, at least, the principal reason—for selecting an extremity is that in anthrax very soon after death a rapid invasion of the blood takes place by putrefactive organisms resembling the *Bacillus anthracis*, and the extremities are the last points to be reached by these organisms; therefore it follows the length of time the animal has been dead before the smear is taken is greatly important. If this is more than twenty-four hours, the microscopical examination will be unreliable.

To assist the bacteriologist in arriving at a correct diagnosis, the stockowner, when forwarding the smear, should include in his details of the death the length of time the animal has been dead where possible, or if he does not know he should say so.

Methods of Infection.—Anthrax is usually confined to certain localities. Moist, marshy places, or soils that are periodically inundated, are particularly favourable to the growth of the organism, especially when the temperature is high. In many such places the disease appears annually, with a variable virulence, principally amongst animals at pasture in the summer-time. When cases occur amongst stabled animals it is usually due to contaminated food. Many of such cases that occur in England are believed—and the evidence is strongly in favour of the view—to be due to material brought in from countries where anthrax is very prevalent.

Cases of sporadic anthrax are extremely rare, if they ever occur. The only common natural method of infection is by ingestion. It is doubtful if even infection by inhalation occurs, and it is only very exceptional that infection by inoculation occurs under natural conditions, although a tiny scratch is sufficient to convey the infection of anthrax.

Treatment.—No curative medicinal treatment is known for the disease; prevention is the thing to be aimed at.

The first broad lines of preventive treatment are perhaps those between different countries. Most countries have the disease to a great or less extent, but those countries that control animal diseases as effectively as possible are beginning to raise barriers against materials known to convey anthrax from countries where the disease is rife or out of hand.

It is a wise proceeding for the individual stockowner to be likewise cautious in the materials that he imports on to his own property. Perhaps one of the readiest things to introduce infection is bone-meal that has not been submitted to a sufficiently high temperature; manure is another. Animals, hides, and skins are also dangerous; riems have been accused of transporting the disease; food for animals will undoubtedly do so.

Next in importance to keeping the disease off the farm is, if the disease should occur, to contaminate the soil as little as possible by the dead carcase. It must be remembered that the blood and internal organs of animals dying of anthrax contain enormous numbers of the organism causing the disease, and when the carcase of an infected animal is opened, not only these organisms are liberated, but they form spores which retain their vitality for long periods, and contaminate the veld upon which they lodge, so that other animals grazing over the ground, even long afterwards, are exposed to the danger of infection. For this reason, where there is the least suspicion of anthrax, such as an animal dying suddenly and without apparent reason, stockowners should refrain from opening or skinning the carcase, but bury it intact after taking a blood-smear for examination. In burying such an animal, it should be buried at a depth of not less than 6 feet, preferably in quicklime, as near the place where it died as possible, but away from any spot where it might contaminate a water-supply; and after burial, if the report received of the result of the examination of the blood-smear shows that the disease has been anthrax, the grave should be bushed over or enclosed in a rough fence to prevent animals from grazing over it. In handling such a carcase, great care should be taken not to allow any discharge from it to get on the clothes or person of anyone touching it, and the ground where the animal has lain should be soaked in a disinfectant, such as a 10 per cent. solution of Jeyes' fluid, and any discharge should be scraped up and thrown into the grave in which the animal is buried, while the hands should be thoroughly washed with disinfectant.

In those places where wood is plentiful the most effectual way is

undoubtedly to cremate (see Disposal of Carcases) the carcase, and with it all material that may have become contaminated by any discharge from the animal. If this is thoroughly done, it does away with all risk of future infection at the spot where the beast died.

Protective Inoculation.—After an animal has recovered from a naturally acquired case of anthrax it is left with a high degree of immunity; this immunity is greater the nearer the animal was to the point of death. Recoveries take place in a considerable number of natural cases in the ox.

It occurred to Pasteur, therefore, to inoculate with attenuated anthrax bacilli.

The Pasteurian Method of Inoculation for Anthrax.—This consists of two inoculations with two vaccines of different strengths, with an interval of twelve to fourteen days between the inoculations. Each vaccine is a broth culture of non-sporulating attenuated bacilli.

The first vaccine is very much attenuated, and is supposed to be devoid of any danger even in large doses for ordinary farm animals. Its purpose is to prepare for the second vaccine, which confers the real immunity.

Bacilli are attenuated by cultivation in broth at a temperature unfavourable to growth—that is, a temperature between 42° and 43° C. The nearer it is kept to 42° the better, but it must not fall below, otherwise the bacilli will sporulate, and it is impossible to attenuate spores. The temperature must not exceed 43° C., because incubation at a higher temperature is fatal to the bacilli.

Pasteur found that by stopping the incubation from the fifteenth to twentieth day a suitable first vaccine resulted, a small dose of which will kill a mouse, but not a guinea-pig or rabbit; and that an incubation of ten or twelve days was suitable for the second vaccine, which will kill guinea-pigs and mice, but not a rabbit or rat.

In practice it is not customary to prepare each batch of vaccine by incubating this way, and the process has only been described to show what a tedious and delicate one it is, and how easy it is for errors in preparation to creep in.

The full immunising effects are not obtained until a week after the second inoculation, and it is probable that during this time the animal has an increased susceptibility to the disease, owing to the natural protective powers of resistance being depressed.

With the inoculation there is no risk of causing anthrax in cattle and horses, but there is a risk in sheep.

It can be shown experimentally, by testing vaccinated and unvaccinated animals, that this method confers a high degree of immunity. . The immunity is strongest the second week after the second inoculation, and ceases to be serviceable in less than nine months. In addition to the risk attending the preparation of these vaccines—in fact, many men refuse to do such work—there are also objections to the method, amongst which mention may be made of the following: It is impossible to obtain a serviceable immunity in less than two or three weeks; the strength of the vaccines cannot be regulated with precision; irregularity in attenuation may cause serious accidents to follow vaccination, especially in sheep, as many as 10 per cent. having died from inoculation; it upsets milch-cows, stops the flow of milk, and the milk is not safe to use as a human food until a week after the second inoculation. Cases in which accidents happen make a serious addition to the pre-existing ground contamination by the urine and the fæces, as well as by the carcase of the animal.

Serum Inoculation.—A protective serum can be obtained by the repeated inoculation of increasing quantities of anthrax cultures. This was discovered by Sclavo, for human beings suffering from malignant pustule, by the hyperimmunisation of sheep.

Sobernheim in Germany prepared for farm animals in this way serum obtained from a horse.

A horse is inoculated by the Pasteurian method, then repeatedly inoculated for some months with increasing quantities of anthrax blood, until finally the horse can tolerate as much as a litre. When this stage has been reached, the animal's serum is then highly protective.

The immunity is acquired the same day that the serum is injected, but has gone the second week.

Sobernheim recommended a mixed method—*i.e.*, to give the animal a dose of serum and at the same time to give a dose of attenuated bacilli corresponding to Pasteur's second vaccine. The serum removes the risk of inoculation anthrax and gives immediate immunity, which lasts during the period that the attenuated culture is working out its active immunity that lasts for months.

This method has been used on a great scale in South America, with good results.

It is alleged that good serum has been successfully employed for

curative purposes, and that two or three doses of 20 c.c. will suffice if applied before the animal is moribund.

Another method recently tried, with encouraging results, in the United States of America is that of conferring immunity by the use of a spore vaccine, which is injected simultaneously with a dose of serum. The advantages claimed for this method are that the vaccine keeps better and immunity is established more rapidly than by the ordinary Pasteur method.

GLANDERS

Synonyms.—Farcy (French, *Morve ;* Dutch, *Droes*).

History.—Glanders was recognised by Hippocrates about 450 B.C., and by Aristotle a century later. The Latin name of it, *malleus*, derived from the Greek, meaning "bad disease or epidemic," owes its origin to the latter writer.

In 1618 the celebrated anatomist Ruini described glanders, and declared it to be "a general disease and incurable." In 1734 Gaspard de Sannier, a French veterinarian, recognised glanders as "highly contagious," and traced the methods of its transmission accurately, but unfortunately others held different views. I have even heard men who claimed to be able to cure the disease.

It may be said little or no progress was made in the knowledge of the nature of the disease until the causal organism was discovered in 1882, and attempts to eradicate it from studs was a matter of great difficulty, almost amounting to impossibility, until the discovery of mallein by the Russian veterinary surgeons Helman and Kalning in 1891.

Glanders has been distributed by trade or war to every country in the world. I am unaware if any records exist of its introduction into South Africa, but one would imagine it would be introduced into the country fairly early in its history. From personal knowledge, I can state that it certainly existed to a considerable extent in many places in the Union of South Africa, as well as in Rhodesia, prior to the Boer War, which is the date some writers appear to regard as its introduction; but, as is well known, war supplies one of the most favourable conditions for the spread of the disease, and it certainly was greatly in evidence during that war.

I landed in South Africa in June, 1896, and had not been many weeks in the country before I came across several cases of the disease, and was able to confirm my diagnosis by application of the mallein test with mallein imported by me from London.

The mallein test in those days was very new, and few veterinarians in South Africa had had previous experience of its employment. I went to Rhodesia in 1897, and found many cases of

glanders soon after my arrival; and as it was on my advice and recommendation that measures were taken to eradicate the disease, perhaps I may be forgiven if I deal rather fully with the history of the disease in that territory. Before doing so I would point out that mankind has to pay a heavy penalty through handling glandered animals. During the year 1891 to 1892 no less than seven European scientists working at glanders lost their lives through accidental infection.

Glanders is almost invariably present amongst equines during the time of war, and it may be that it was owing to the native rebellion of 1896 that the disease was so prevalent in Rhodesia during the two following years. In 1898, following an agitation by horse-owners, the Administration decided upon taking steps to eradicate the disease from the territory, and to that end, after publication of the High Commissioner's proclamation No. 2 of January 5, 1899, authorising action, Government Notice No. 100 of April 13 was promulgated, which aimed at, by applying the mallein test to all horses, mules, and donkeys in the country, likewise to all such animals entering the territory, not only freeing the country from the disease, but also to keep it free. It was a fairly formidable proposition to undertake, and, so far as I am aware, no other territory had at that time undertaken such a task; but the conditions prevailing lent themselves to it, and the result that has been obtained cannot be considered other than most satisfactory.

At the start a great number of animals had to be destroyed—in fact, practically all the horses belonging to the British South Africa Police and many of their mules were shot. Naturally, with the introduction of something so new to horse-owners as the mallein test, a certain latitude had to be given with respect to animals that reacted to the test, but were not visibly affected with the disease; and in 1904, at a Veterinary Conference held at Bloemfontein, the Chief Government Surgeon of the Cape Colony, in advocating the adoption of the mallein test, recommended that animals responding to this test might be isolated and not destroyed until visible symptoms manifested themselves. This was an adoption of the method that had originated in Rhodesia, where owners soon came to appreciate the reliability of the test, and if an animal reacted to it they were anxious for its destruction.

Some little time elapsed before all the animals in the outside districts made their way into the towns, where they were subjected to the test, and eventually the country was freed of the disease; but, as might be expected, a few recrudescences of the disease

occurred, perhaps due to faulty application or bad mallein. In 1910, after three years' freedom from the disease, outbreaks occurred at Salisbury and at Bulawayo; again in 1911 one case of the disease occurred at Gwanda and another at Salisbury. Under a system of following up the animals that had been in contact and applying the mallein test, the outbreaks were quickly cleared up. With regard to the testing of animals entering the country, many animals were found to be infected and were destroyed. Cases were plentiful in the year following the Boer War; they have gradually decreased since that time, until now it is rare to find a case amongst imported animals. No case has been found in animals within the country since 1911.

Nature.—Glanders is a specific disease of a malignant character, due to the growth and development within the animal body of a micro-organism, the *Bacillus mallei*, discovered by Löffler and Schutz in 1882.

Glanders is mainly a disease of equines. The continued existence of the *Bacillus mallei* is bound up with the continued existence of the equine species. Cases in man or other animals represent an overflow or accidental infection from the equine species.

In natural circumstances cases occasionally occur in the cat; it has also been said to occur in the goat, also in wild carnivora kept in zoos and menageries, and a considerable number in human beings. Natural cases have never been seen in the ox, and this animal has a nearly absolute natural immunity to the disease; it is almost impossible to infect it. The pig and the dog also possess a considerable immunity, but not so great as the ox.

With regard to the susceptibility of the horse, mule, and donkey, some veterinary writers have stated that the donkey is very susceptible to the disease—more so than the horse. My experience has led me to quite a contrary conclusion, and I place the degree of susceptibility in the order in which the animals are mentioned. I may claim that my experience of this disease has been extensive, and perhaps unique, so far as donkeys are concerned. I have seen scores of donkeys running loose amongst horses and mules in large stables in which cases of glanders have been present, and by other ways possessing every opportunity of contracting the disease, and this at a time when many horses and a small number of mules were destroyed on account of the disease. I also have mallein-tested and had tested under my supervision several thousands of donkeys, and have never discovered during my twenty odd years' experience a case of glanders

amongst these animals; therefore I am forced to the conclusion that donkeys possess a considerable degree of immunity and are less susceptible to the disease than mules, and much less so than horses.

In the past it was customary to speak of glanders and farcy as two diseases. The former was the term used when the disease manifested itself in the nostrils and lungs, and the latter when it manifested itself in the skin. No good purpose is served by continuing this distinction; both manifestations may occur in the same animal, and as both are caused by the same organism, the tendency at the present time is to drop the term farcy. With regard to the nostril and lung manifestation of the disease, two forms occur, the acute and the chronic; therefore, in considering the symptoms, it may be advisable to detail the conditions prevailing with all three manifestations.

Symptoms in Acute Glanders.—The first symptoms noticeable are: the animal neglects its food, is feverish; the glands under the jaw are swollen; there is a discharge from the nose, usually from both nostrils; the membrane lining the nostrils is of a dark red colour; and in the course of a day or two small ulcers will be seen in this membrane. They are usually about the size of a pea, and look very much as if they had been cut out with a saddler's punch. In many cases it is necessary to wipe the dried discharge off before one can see this characteristic ulceration of the membrane. A discharge from the nose, tinged with blood, is always most suspicious of ulceration being present; perhaps it may be higher up than one can see with the naked eye. Swollen nostrils, and often a roaring sound in breathing, are caused by the thickened membrane interfering with respiration. The termination of a case of this description depends on the strength of the animal and the condition of its vital organs. It may terminate fatally in a few days, or the constitutional disturbance gradually subsides; the discharge from the nose becomes less, but as a rule never entirely disappears, although it may become intermittent; the glands become smaller and the ulcers slowly heal, leaving a white scar. This apparent recovery is in the majority of cases unreal, for the poison may not leave the system, but may quietly develop and increase in the lungs. Such a case in this stage would be considered as one of chronic glanders.

Symptoms in Chronic Glanders.—It is not necessary for the production of chronic glanders that the acute form of the disease shall have preceded it. In the majority of cases—probably 90 per cent.—the disease gradually develops, the first symptom

often being a discharge from the nose, usually from one nostril only, with the gland under that side of the jaw enlarged to about the size of a walnut, but it may be larger or smaller. It feels lumpy and nodulated, not smooth and round; it remains pretty firmly fixed to the jaw, keeps hard and the same size, and does not show any tendency to gather and burst like an abscess such as is seen in strangles. It is very rare that the glands gather and burst in glanders. The discharge from a horse's nose in glanders may be little different to that seen in an ordinary cold or strangles, but it is extremely sticky, and frequently adheres to the front legs and other parts of the body; the membrane lining the nasal chamber is altered in colour, being a pale blue grey. This is followed by the characteristic ulceration. An animal suffering from this form of glanders has an unthrifty appearance, often a profuse urinary discharge, appetite abnormally large, but does not improve in condition; frequently there is swelling of the legs, often a short hollow cough, and a gentle punch in the ribs may cause the animal to grunt, indicating that the lungs are affected.

Symptoms of the Skin Form of the Disease or Farcy.—The first symptom is usually a swelling of one or other of the limbs. In a short time characteristic little hard swellings (farcy buds) about the size of a Barcelona nut appear, with cords radiating outwards and from one swelling to another. These cords look very much like a weal caused by a whip-lash. Usually in two or three days (it may be a week, according to the severity of the case) the swellings burst, and discharge rather thin matter tinged with blood, which dries on the hair, forming a yellowish crust. It is common to get larger swellings without cords, about the size of a hen's egg or orange, on the back and hind quarters. If much constitutional disturbance and fever are present the disease assumes an acute form, the buds and cords extending over a greater part of the body. The buds, bursting in quick succession and discharging pus, give the animal a loathsome appearance. Farcy buds may appear on any part of the body, but are more frequently seen on the limbs, face, neck, shoulders, and sides.

Post-mortem Appearances.—The opening up of a glandered carcase is an operation that should be conducted with care, although slaughtermen and others take few or no precautions. If the skull is carefully split up a good demonstration of the ulceration before described may frequently be obtained, but the lungs are the organs in which typical lesions of glanders are frequently found. These consist of small nodules varying in size from a grain of kaffir corn to a pea, glassy and pearl-grey in colour,

and when cut into a pale yellow speck is frequently found in the centre of them. In a smaller number of cases what is termed "infiltrated glanders of the lungs" is present, somewhat resembling a tumour within the lung tissue, dirty white in colour, irregular in shape, the size of an orange or larger.

Diagnosis.—Ulceration of the nostrils and other symptoms, when present, are a sufficient diagnosis, but the trouble used to be to diagnose a case before such typical symptoms presented themselves. As a young practitioner in London, I frequently adopted the practice of administering a pretty strong physic ball, and by thus reducing the animal vitality the typical symptoms appeared. There is no necessity for such expedients at the present time, as the mallein test for the disease is, in my opinion, as nearly infallible as it is possible for such a test to be. The incubation of naturally contracted glanders is from one to three months before typical symptoms are shown, and it is during this period that mallein has proved such a boon and assisted so greatly in the eradication of the disease. Other means of diagnosis are by the microscopical demonstration of the organism, by inoculation of susceptible animals, and by the cultivation of the organism.

At the present time there appears to be no necessity to describe the mallein test; full instructions as to its method of application and the reaction following are sent out invariably with the material.

Treatment.—No one claims or attempts to cure glanders at the present day, although such attempts were common in the past, and perhaps some may have had a certain amount of justice in their claims, in that a small percentage of animals do undoubtedly recover from glanders, whether they are assisted by man or otherwise; but the economic thing to do is to eradicate the disease by slaughter and disinfection.

CONTAGIOUS ABORTION IN CATTLE

Synonyms.—Epizootic Abortion, Infectious Abortion.

History.—The records of the disease are lost in antiquity, whilst its infectious nature has been recognised from very early times. Mention is made of it in Genesis xxxi. 38, and old English writers on stock were greatly of the opinion that the malady was a contagious one, and advised treatment accordingly; but it was not until 1896 that the organism causing the disease was discovered by Bang, a Danish veterinarian. This was known for some time as Bang's bacillus, but is now usually called *Bacillus abortus*.

The disease is of world-wide occurrence, is one of the most serious diseases of stock, and causes enormous losses in many parts of the world. In Great Britain it is regarded as second only in importance to tuberculosis, while in the United States of America the exact financial loss cannot even be approximately estimated; but from the fact that the disease exists in all sections of the country, both in dairy and range cattle, it can safely be stated that the direct loss reaches into millions, while the potential loss is likewise enormous and inestimable. In New Zealand the disease costs the country a considerable loss, and the same remark applies to a lesser extent to the Union of South Africa, where the disease has played sad havoc in many herds of cattle. Rhodesia up to the present has been more fortunate in the number of outbreaks occurring. Mr. Bevan, the Government Veterinary Bacteriologist, by means of the agglutination test diagnosed the disease in a herd of cattle at Sipolilo, and by comparative tests came to the conclusion, as stated in his annual report of 1914, "that the specific contagious abortions of those countries—namely, Great Britain, British East Africa, Union of South Africa, and Rhodesia—are identical. Samples of the same blood were sent to Sir John MacFadyean, of the Royal Veterinary College, London, who was able to confirm the diagnosis." This was, I believe, the first scientific finding on the point emanating from South Africa.

This is a matter of great importance in view of the research work that is being carried out in connection with the disease

in different continents, as it enables the findings of one investigator to be verified by another in a different part of the world.

The disease was first discovered in Rhodesia in October, 1914, and since that date several other outbreaks have occurred in the eastern portion of that territory; but up to the present the existence of the disease has not been diagnosed in the western portion. The occurrence of the disease there opens up a pretty problem to speculate upon as to whether it has been prevalent, but unrecognised for a longer time, or whether it is a new importation. There appears to be some foundation for the belief that the disease existed in the country, and was known to the natives prior to the European occupation. Time may prove which of these theories is the correct one, as well as perhaps disprove some of the theories that are at present held in regard to the disease. For it must not be forgotten that there is still much to be learnt in connection with it, and a successful method of handling has still to be found.

Nature of the Disease.—Abortion is a peculiar disease, in that it seems not to affect the general health of the mother; but the germs, gaining entrance to the pregnant uterus, set up an inflammatory process which breaks down the attachment between mother and offspring. The latter, unable to receive either nourishment or oxygen, dies, whereupon it becomes a foreign body and must be expelled. The act of abortion is but a symptom of the disease, and may occur at any time during the period of pregnancy. Young animals in first pregnancy usually abort early, at from two to four months, while older cows may carry calves five to eight months or even full time. The greatest number of cows abort in the seventh month. One observer found that 50·5 per cent. of the animals that came under his observation did so. Abortion may occur in susceptible cows at any period of their lives from first pregnancy to fifteen or more years of age.

Investigators have been able to produce abortion experimentally in various species of animals by inoculating them with the abortion organism, but under natural conditions the disease does not readily spread from one species to another, as from the cow to the mare; and it has been held that abortion is produced in these species of animals by three different organisms, there being one for the cow, another distinct germ for the mare, and still another for the ewe.

A point that elicits a great amount of enquiry is the duration of the disease and the number of animals in a herd that are infected. In this, as in all other diseases, a certain number of

animals are found resistant to the disease and do not abort, although they may be infected. Investigation has shown that approximately two-thirds of the cows that abort lose their calves but once, and thereafter become immune and calve normally. About one-third abort a second time, and only a small percentage a third time.

The complications which attend the disease are more serious than the disease itself. Retained afterbirth, barrenness, the loss of milk in dairy herds, and the lessened breeding efficiency in pure-bred and beef herds, are among the greatest of losses; also calves may be born alive, but so weakened because of the disease that they quickly die of other ailments or do not thrive.

Symptoms.—The symptoms of approaching abortion are merely those preceding normal calving, and consist in the swelling of the udder and the external genital organs. The swelling of the udder will be more apparent with beef cows and heifers than with cows in milk. Not infrequently the calf is expelled without any warning symptoms. In very early abortions the calf and its membranes are expelled intact. In such cases the calf is often not found, there are no observable symptoms, and no disturbance in the health of the cow; consequently the owner thinks the cow has failed to breed. Frequently abortion is preceded by an odourless discharge, and is followed by a dirty yellowish to brownish flaky discharge that in turn gives place in a few days to a purulent discharge, which may persist for several weeks. In addition, there are certain inflammatory changes present in the body of the dead calf, notably in the fourth stomach and intestines, and a gelatinous infiltration is found under the skin, also the fœtal membranes are covered with a fibrinous purulent exudate; but owing to the frequent disappearance of both the dead calf and its membranes, it may be said that really no clinical symptoms exist upon which a reliable diagnosis can be formed.

Diagnosis.—Various ways, based upon the presence of the bacillus, are employed for diagnosing the disease. Perhaps the simplest way is by demonstrating the bacillus itself under the microscope. To do this a smear from the mother or from the exudate that is present and adhering to the membranes of the calf is taken and stained for examination in the usual way. Another way is by the experimental inoculation of smaller animals, such as the guinea-pig; but the method that is relied upon to the greatest extent of all is what is known as the agglutination test, which is a specific reaction between the serum of the infected animal and the *Bacilli abortus*.

In his report Mr. Bevan states regarding the agglutination test: "A method whereby blood can be collected in pipettes containing a certain quantity of preservative was finally adopted, and has proved highly satisfactory in practice and so simple in application that the samples can be taken by stockowners themselves." So no obstacle is in the way of an owner finding out if he has the disease in his herd or not.

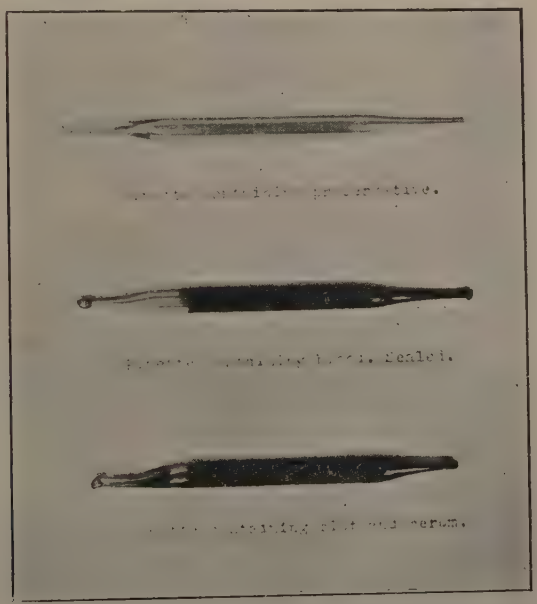


FIG. 4.—BLOOD COLLECTION FOR THE AGGLUTINATION TEST.

Methods of Infection.—The disease may be experimentally conveyed to a susceptible animal in a number of different ways, but the natural means of infection is believed to be in almost all cases by way of the contaminated food or water or through the bull. The organism may possibly, in rare cases, obtain entrance to the body through the external genitals, also through the teats into the udder, but probably such methods of infection are so rare as to be unworthy of practical consideration.

Of the two methods mentioned, for a long time the bull was regarded as the most potent source of infection, but latterly research points to this being an error, some investigators contending that bulls are not infected with the disease for a great length of time. Others, again, hold opposite veiws. What

apparently happens is that a certain number of bulls recover from the disease, and are no longer a source of infection and danger when used in a healthy herd of cows. In other bulls the disease may become located in the genitals, when they remain a source of infection; but unless this occurs, the evidence appears to be in favour of the bull's body not acting as a reservoir of the disease. Of course, the mechanical transmission of the disease by the bull from an infected to a healthy cow is always possible, and may be a prolific method of spreading the disease in an infected herd.

FIG. 5.—METHOD OF COLLECTING BLOOD FOR THE AGGLUTINATION TEST. (Photo by Bevan.)

It is apparently accepted by the majority of investigators now that the disease is contracted more frequently by ingestion than in any other manner, and when it is remembered how easily the food of healthy animals can be contaminated by the discharges from a diseased animal the theory appears to be a common-sense one. The various ways this can be brought about will occur to all. To take a few, in stabled cattle the feet of the attendants prove a ready source of conveying the infection from contaminated bedding to the food of healthy animals; at pasture the feet

of the grazing animals would tend to distribute the infection about the pasture; cows drinking in a dam might also prove to be a very dangerous source of infection. Infection is doubtless most frequently conveyed to a healthy herd by an apparently normal but infected female.

Probably the most interesting fact that has recently been found out in connection with the disease is the discovery by Messrs. Shroeder and Cotton, two American scientists, that the udder is the reservoir of the disease. This discovery was made quite accidentally by the experimental inoculation of market milk into guinea-pigs, producing lesions typical of the *Bacillus abortus*. Upon further research being made, an animal was found which had the organisms present in the milk continuously for seven years. Further work to prove the finding was done, and much that was obscure before was cleared up. A further interesting fact was established that in an infected cow that has returned again to normal parturition the afterbirth may still be infected.

Treatment.—In the present state of our knowledge we cannot truthfully say that the disease can be eradicated. Experimenters have been groping about for cures, but there is no cure. A few years back carbolic acid was acclaimed a cure, the method adopted being to progressively dose the animal until its system became saturated with the drug. Methylene blue was another. There were others, but after trial they all had to be discarded as worthless. Upon the discovery of the agglutination test the method of sorting the infected from the healthy and keeping them in separate herds suggested itself, but if this method is scientifically sound—a matter in which a certain amount of doubt is excusable —the carrying of it out in most or many cases is a practicable impossibility. Vaccines of both living and dead organisms have been tried with varying results, and are perhaps the most promising line of treatment that has been so far tried; but until science comes further to his aid, the unfortunate owner of an infected herd can only do his best to control the disease and develop the immunity of his herd.

The dead calf and its membranes and accompanying discharges are teeming with the germs of the disease. They should be promptly gathered up and destroyed by burning or buried deeply. The aborting cow continues to disseminate infection for weeks, and far more disease is spread at this time than at any other; therefore the cow should be isolated and not put to the bull for some time. Two months is about the shortest time in which the uterus becomes free of the disease; so to be on the safe

side, allow another month or two. The quarters that the cow is isolated in should be frequently disinfected and whitewashed, and the litter and everything else that may have become contaminated with the discharges must also receive attention.

It may sound a heresy to advise an owner to develop herd immunity, but in the majority of cases this will be the best thing to do. In many cases the disease will have got such a hold on the herd before it is diagnosed that no other method will be available, and what cannot be cured must be endured. If an owner decides to follow this line of action, he will never sell a breeding animal, cow or bull, on account of the disease. To use the South African expression, such "salted" animals will be amongst his most valued possessions. The law of the survival of the fittest holds here. He will not introduce fresh females into the herd if he can avoid it, because such a proceeding is often like adding fresh fuel to a fire that is gradually dying out—it makes it flare up again.

The next line of treatment is in connection with the infected animal, and to prevent the mechanical transmission of the disease by the bull the long hairs around the opening of the sheath should be clipped off, and the surrounding surface scrubbed with soap and disinfectant. Then the interior of the sheath should be irrigated out with a weak disinfectant, such as a teaspoonful of carbolic acid to a quart of water. Close the orifice by pressing with the fingers, and work the disinfecting solution about with the other hand.

With regard to the cow, it is better in the majority of cases to leave the cure of the womb to nature, and simply wash the discharges from the outside parts, tail, and legs with disinfectant; but retention of the afterbirth is a frequent sequel to abortion. In such cases interference is advisable, and a carbolic solution, as advised for the bull, may be used to wash out the uterus. But even that may cause severe straining, and if it does it is better to use a solution of one level teaspoonful of salt to a pint of tepid water. If much decomposition and stench is taking place, a gelatine capsule containing ½ to 1 ounce of iodoform may be inserted into the uterus, where it will dissolve.

CONTAGIOUS BOVINE PLEURO-PNEUMONIA

Synonyms.—Pleuro-pneumonia Contagiosa, Lung Plague of Cattle (French, *Péripneumonie contagieuse;* German, *Lungenseuche Viehseuche;* South Africa, Lung-sickness; Matabele, Ndaliman).

History.—The first authentic information of this disease was at the end of the seventeenth and beginning of the eighteenth centuries, when it was raging as an epizootic in Switzerland and the adjoining territories, from whence it extended to all countries of Western Europe, and from them, by the transportation of cattle, it spread to far-distant lands over the seas. In 1839 it was introduced into Ireland as the result of the importation of some Dutch cows from The Hague. In 1842 Great Britain was infected by cattle from the Continent. In 1843 the United States of America and in 1859 Australia were similarly infected.

The disease was introduced into Cape Colony in 1854 by some Friesland bulls which were imported from Holland and landed at Mossel Bay. From that date it has spread all over South Africa, and has been the cause of serious annual losses of cattle in the sub-continent.

In 1861 it reached as far inland as Matabeleland, and caused heavy losses in this at that time heavily stocked country. Umziligasi, the King of the Amandibele nation, called to his assistance in dealing with the plague Messrs. Moffat, Sykes, and Thomas, of the London Missionary Society, who were resident in the territory, and inoculation was practised; but the disease destroyed many cattle and became enzootic in the country, so when the Matabele, after their periodic raids into surrounding territories, returned with numbers of loot cattle a considerable proportion of these animals contracted the disease and died.

In 1893, when Matabeleland was occupied by the British South Africa Company, and during the following years, the disease was very prevalent. Rinderpest in 1896 destroyed many lung-sickness infected cattle, but the disease survived the ordeal; and when the country was again being restocked after rinderpest had swept through and died out, lung-sickness again came prominently

into view and destroyed many of the recently imported cattle.

In 1901 lung-sickness was present to a great extent; but the introduction of East Coast fever took place that year, and during the following years the spread of the latter disease destroyed a great number of lung-sickness cattle, and finally eradicated the disease in about 1903 or 1904, since when the disease has not again appeared in Rhodesia.

The position in the Union is nearly as satisfactory. During the last decade strenuous efforts have been made to eradicate the pest, and the result attained may be regarded as most satisfactory, as during the last five years no cases have occurred.

Unfortunately such a favourable state of the disease cannot be recorded in the Bechuanaland Protectorate. Great strides in getting the disease under were being made in that territory, but in 1918, owing to the epizootic of human influenza placing the majority of the inhabitants *hors de combat*, and so disorganising the control that was being maintained over the disease, a severe spread of the disease occurred into parts of that territory in which it had previously been eradicated.

Other places in South Africa where the disease is present are Ovamboland and the extreme north of the south-west Protectorate, close to the Ovamboland border.

Nature.—Lung-sickness is a specific infective disease caused by a bacterium that is on the border-line of invisibility, as it is so small that it is only seen as a point when magnified 1,500 times. The disease is peculiar to the bovine species, and is not seen or transmissible to any other animals. It is ordinarily contracted by cohabitation or immediate contact of healthy and diseased animals, but nothing very certain is known regarding the natural method of infection; it is believed that infection is transmitted by inhalation and by dust.

Before any symptoms are observed an infected animal can spread the disease, but when the disease is at its height the infection is most virulent.

The Period of Incubation.—This is usually stated to be from three to six weeks. In exceptional cases visible symptoms may develop earlier, and there is little doubt that in some mild or suppressed cases three or four months may elapse before symptoms are observed.

The Animals' Diseases Consolidation Ordinance of 1904 (Rhodesia) imposes a special warranty upon the vendor of cattle, and if the disease breaks out in such cattle within twenty-one

days after purchase, and also if it spreads from such animals to the herd of the purchaser, the seller can be held responsible for all damage done. The warranty also implies that cattle sold by public auction or private treaty have not been inoculated less than sixty days. The Ordinance also prohibits the importation of cattle infected with lung-sickness or that have been inoculated within sixty days, and in addition to the usual penalty of fine or imprisonment for contravention, the importer is held liable for all damages caused by infection from such cattle occurring within thirty days after arrival.

The Diseases of Stock Act, 1911, of the Union, which is drafted on similar lines to the Rhodesian Ordinance, does not specifically lay down the foregoing conditions; but Roman-Dutch law, which is in force in both territories, implies a very similar warranty under what are regarded as latent diseases and other defects.

Symptoms.—The first symptom presented is a dry, husky cough, which gradually increases in frequency and intensity, and becomes very painful, causing the animal to stand with its front legs wide apart, arch the back, extend the head, and protrude the tongue during the act of coughing. The breathing becomes accelerated and is accompanied by a grunt; the animal is disinclined to move or lie down owing to the pain caused by doing either; the appetite and rumination disappear, accompanied by a rapid loss of flesh; and as more of the lung tissues become involved, the affected animal gasps for breath with the mouth open, the nostrils dilated and quivering, and the muzzle covered with frothy saliva.

During the progress of these symptoms in quiet animals, by auscultation and percussion it is possible to follow the changes that are occurring in the lungs, usually only in one lung. The pleurisy is denoted by pain on pressure between the ribs; the consolidation in the lung results in loss of sound; and the exudation of fluid into the chest cavity is indicated by the well-marked line of the dull sound upon percussion up to the level to which the fluid has reached.

The bowels are at first usually constipated, but this is frequently followed by diarrhœa, and the animals are subject to tympanites, especially after drinking.

Post-mortem Appearances.—On opening the chest cavity a quantity of yellowish turbid fluid is found on the affected side; floating in this are found clots of fibrin resembling the white of a poached egg. The surface of the lung and the ribs are covered with a similar material, and the lung is frequently found adhering

to the ribs by this material having become organised, the strength of the adhesion depending upon the length of the time it has been present.

The affected portions of the lung are solid, heavy, sodden, and stand out from the other portions. On cutting into them, the section presents a marbled appearance, portions of which will be found to be much darker in colour than others; usually a quantity of lymph exudes from the white thickened septa between the lobules of the lungs.

The foregoing appearances are found in animals that are destroyed soon after they have become visibly infected or in animals that have died within a month of showing symptoms. In animals that have lived longer the parts may become more purulent and emit a very offensive odour when the chest is opened; they also become drier with less fluid present, and in "lungers," or those animals that have partially or totally recovered from the disease, the affected portion of lung may have assumed more of the appearance of an abscess enclosed in a fibrous capsule.

Diagnosis.—A correct diagnosis of an isolated case of lung-sickness cannot with certainty be made in the living animal; a post-mortem examination to corroborate the symptoms presented is essential.

Naturally the case is different when the disease is known to exist in a herd or in the locality; then it is possible to give an opinion with more assurance even in the earlier stages of the disease. A favourite and a valuable aid to diagnosis is to drive the suspected herd around swiftly, when, if the disease is present, one or more animals will evince the cough as described.

Treatment.—The treatment of the disease hitherto adopted in South Africa has been that of isolation, quarantine, and inoculation, or drenching with the fluid obtained from the lung cavity.

Destruction of all visibly infected animals was not always carried out, but little hope of eradicating the disease by this method can be entertained unless this is done.

D. Hutcheon, M.R.C.V.S., late Chief Veterinary Surgeon and Director of Agriculture, Cape Colony, had a very large and extended experience of this disease and of this method of combating it. The following extracts are taken from an article written by him in 1905:

Method of selecting the Virus.—An animal suffering from the disease in its earlier stages should be selected by preference. It should be killed and properly bled. The carcase is then laid on its back and the skin removed from both sides of the chest.

The breast-bone and costal cartilages are then removed. This will expose the lungs, and show on which side of the chest the pleural fluid is contained, if there is any present. This fluid may be either scooped out with a small jug, or a tube may be inserted between the ribs, as near to the spine as possible, and the fluid drawn off into a flat basin.

If this fluid is bright, clear, and thoroughly aseptic, it may be used for inoculating with, but if it is muddy, septic, or blood-stained, it should not be used for inoculation. It is necessary then to obtain the virus from the lungs. Remove the whole of the diseased lung carefully from the chest, place it on a clean table or sheet of corrugated iron in a cool place. Let it lie for half an hour or more until the blood has had time to coagulate; then disinfect the surface of the lung, and with a sharp-pointed knife carefully make an incision along the white bands of inter-lobular tissue, converting them into a sort of open drains; the serous fluid, or lymph, will then gravitate into these channels, and enable you to scoop it out with a narrow teaspoon or eggspoon, or it may be drawn up with a syringe.

Another method is to carefully separate the pleural covering from the diseased portion of lung from which you are to extract the virus in such a way as to form a pocket at the lower border of the lung; then open the lymph bands as above directed, and allow the virus to drain into this pocket, from which it can easily be lifted out with a spoon and transferred to a wide-mouthed bottle or jar. Still another method recommended is: "To dig one or several funnel-shaped holes in the diseased tissue, into which the liquid will percolate, and thence be scooped out" (Theiler).

In the more recently affected portion of the lung the spaces between the interlobular lymph bands present a pinkish-yellow colour, and its cells are filled with a clear serous fluid. It is from this recently affected portion that the best virus is obtained, and should always be selected. The virus usually coagulates; immediately after it is withdrawn from the lung it should, therefore, be strained through two or three folds of clean muslin, or other porous cloth, and placed in sterilised bottles, and kept in a cool place. Unless the virus is to be used at once, 1 fluid dram of pure glycerine should be added to every ounce of virus, or 1 part of glycerine to 8 parts of virus, and shaken together. But although the virus can be preserved for a considerable time in this manner, it is advisable to use it within fourteen days; it cannot be depended upon if kept much beyond that period, unless

it can be kept in an ice-chest, when it retains its virulence much longer.

All instruments and utensils used in collecting and preparing the virus should be sterilised.

The Cultivation of Pleuro-Pneumonia Lymph in Young Calves.—Pasteur's assistants, Dr. Germont and M. Loir, carried out a series of carefully conducted experiments for the Queensland Government in 1888 to 1889, with the object of discovering an improved method of inoculation against the contagious pleuro-pneumonia of cattle; and amongst many interesting facts which they elucidated, they proved "that the virus taken from the lung of an animal suffering from pleuro-pneumonia and inoculated behind the shoulder of a calf from five to six months old produces a large œdema, and that the liquid or lymph taken from this œdema has always proved virulent, and that it maintains its virulence after three successive transmissions through calves; but in order to preserve this virulence it is necessary from time to time to rejuvenate the virus by a return to a case of spontaneous pleuro-pneumonia."

The calves destined to furnish the virus should be inoculated in the neck or behind the shoulder—the latter by preference—by the hypodermic injection of about 15 drops of virus obtained from the lung or pleura of an affected animal.

After from five to ten days, or sometimes a little later, a slight swelling is observed, which increases during the following days, both in thickness and in width. This swelling is painful when pressed, and as it expands the temperature of the animal becomes progressively higher, varying from 105° to 107° F.

At the end of one or two weeks the swelling ends by covering the whole of the abdomen, extending sometimes from the groin to the neck. Generally, however, it remains limited to the side of the body on which the inoculation was performed; occasionally the exudation spreads into the thoracic cavity.

The animals do not always succumb to the consequences of inoculation; the chances of survival are all the greater the older the animals, and in some cases the swelling remains circumscribed, becoming no larger than a cricket-ball and fibrous in character. In these cases practically no virus will be obtained.

The death of the calves may be awaited, or they may be slaughtered in the full evolution of the disease, when the œdema is sufficiently large for practical purposes. Before opening the tumour to collect the virus, it is necessary first to break down with a few smart strokes with a hammer or wooden mallet the

meshes of the connective tissues in which the lymph is imprisoned; this facilitates the collection of the virus, and has, moreover, the advantage of mixing the lymph with the solid particles which appear to contain the active portions of the virus.

To collect the virus the body is placed on the healthy side, and an incision is made along the whole extent of the tumour, and the skin is raised in such a manner as to form a pocket, in which the liquid will collect and from which it can easily be extracted. The meshes of the connective tissue may be further lacerated with the knife, if necessary, to free the virus. When the lymph is being collected in this way it is a transparent limpid fluid, but after a short time the fibrin coagulates and forms a clot; it is necessary to inoculate some of this solid matter.

All the operations must be carried out with the utmost cleanliness and antiseptic precautions, the hands and instruments must be disinfected, and the vessels for receiving the virus properly sterilised. When the virus is to be kept, an antiseptic, such as glycerine, 1 to 8 of virus, and $\frac{1}{2}$ per cent. of carbolic acid, is added to preserve it.

Artificial Culture of Pleuro-Pneumonia Virus.—The artificial culture of pleuro-pneumonia virus is made in a medium called "Bouillon Martin," to which about 5 per cent. of normal serum of an ox is added. The culture is easily obtainable by filtering the "virus" taken from the chest of a lung-sick animal, together with the broth—"Bouillon Martin"—in the proportion of about 1 to 2 per cent.

The broth, when kept in the incubator, becomes after two or three days slightly opalescent, which indicates that the microbes are growing. Such a culture, when injected into a susceptible animal, behaves exactly as does the liquid taken direct from the sick animal.

It has the advantage that it can be continuously transplanted into new media for many generations.

These retain their virulency; it is, in fact, pure virus (Dr. Theiler).

Methods of Inoculation.—The farmers practise several methods. A common practice is to make an incision under the skin of the tail and insert a piece of wool which has been soaked in the virus, and maintaining it in position by a string or strip of calico. If the wool were previously cleaned in boiling water and dried in the sun there would be no objection to this method. But, as generally used, the wool is liable to contain septic organisms.

A very objectionable method of inoculation is sometimes

practised by the farmers; it consists in making an opening in the skin of the tail, or cutting a piece of the skin out, and fastening a piece of the diseased lung on to the wound. Septic infection of the wound, resulting in a large mortality, is almost certain to follow this method of inoculating.

The usual method of inoculation practised is the insertion of a seton soaked with virus under the skin of the tail near its point. The instruments required are an inoculating needle, a pair of good-sized sharp scissors—a good sheep-shears acts well—and some woollen thread, such as Berlin wool or Scotch fingering.

The woollen thread should be cut into 4-inch lengths and placed in open-mouthed bottles, which for convenience should hold from 4 to 6 ounces of virus. Several hundred threads can be soaked with the virus in each of these bottles, and they are easily extracted with a forceps as required.

The Inoculating Needle.—The inoculating needle is about 4 inches long, with an eyelet near the point, its base being inserted into a convenient handle.

In securing cattle for inoculation, trained transport oxen may be caught and fastened by the horns to one or two waggons placed lengthwise in front of each other, the oxen being packed together as close as possible. Cows and other tame cattle can be caught by a riem, after which a man at each horn can hold the head, while another fastens a rope or riem round the hind legs to hold them firm. This protects the operator, while it prevents the animal from jumping about. Semi-wild animals must be cast, or passed into a crush.

In thus preparing the way for the operator, great care should be exercised not to harass, beat, or bruise the animals in any way, and on no account should any animal be caught by the tail when being thrown. Any such injury is liable to induce swelling at the part injured when the virus is circulating in the blood.

Having secured the animal, the operator proceeds to clip off the hair from the brush for about 2 inches upwards from the point of the tail on the upper side. The part should then be cleaned and disinfected with a brush and suitable fluid. To facilitate matters, this should be done by the assistant. The operator then holds the tail firmly by the brush with his left hand, while with the right he carefully passes the needle up under the skin in a straight line for 1½ inches, passing the point of the needle out by gentle pressure on the handle, after which he moves the needle so as to enlarge the channel. One saturated thread is then put through the eye of the needle and drawn till both ends are

even. The operator now retracts the needle, drawing the thread into the channel after it, meanwhile gently compressing the lower orifice with the thumb of his left hand to retain the virus. When the point of the needle is clear, the thread is clipped off close to the needle, thus leaving the main portion in the channel. In this manner, with plenty of assistance, one inoculator can inoculate from three to four hundred in a day.

During recent years, more particularly in the Australian Colonies, the needle and thread have been largely replaced by the hypodermic syringe for inoculating cattle as a preventive of the contagious lung-sickness. The method adopted is similar to that now so largely practised in inoculating cattle against quarter evil. The hair is clipped from the upper surface of the tail for about 6 inches, extending from the tip. The part is then cleaned and disinfected with a 5 per cent. solution of carbolic acid or Jeyes' fluid applied with a brush. Then insert the hypodermic needle under the skin, and carefully pass it down between the skin and subcutaneous tissues for about 1½ inches, move the needle from side to side so as to enlarge the channel at the lower end, then withdraw it a little, attach the syringe to the needle, and inject the virus—about 5 or 6 drops—then withdraw the needle and syringe together; at the same time, with the thumb of the left hand, close the puncture in the skin, and gently rub the part containing the virus so as to spread it out in the cellular tissue. In Australia they have hypodermic syringes which regulate the dose from a well which is attached, and the operator simply injects the dose under the skin without any preparation of the part.

A very strong syringe and needle are required, as the skin near the point of the tail in full-grown cattle is firmly attached to the subcutaneous tissue. This method of inoculation has the great advantage that there is no open wound by which septic or other germs can enter.

A pock is expected by many, but this is not usual, nor is it necessary. Theiler says that it should reach at least the size of a hen's egg. It is usually severe, and not without danger if it does. Such a swelling, if left alone, has a great tendency to extend up the lymphatics of the tail to its base, often involving the tissues of the hind quarters and the organs of the pelvic cavity. When the exudation extends up the tail, one of the first indications is swelling of the lymphatic glands or "kernels" on each side of the root of the tail. Many farmers cut these glands out—I have frequently done it myself—and when the operation is performed before the exudation has extended to the surrounding

tissue, it is a very effective means of preventing the further extension of the swelling. "But the swelling of the glands is secondary to the irritation, swelling, and death of the tissues at the point of the tail. Hence, by amputating the tail upwards from the tip until healthy tissue is reached, there is no necessity for touching the glands until they show indications of containing pus, when they should be freely opened; meantime they should be dressed with the following mixture:

Linseed oil	.. .. ..	1½ pints.
Turpentine	.. .. ..	4 ounces.
Carbolic acid	.. .. ..	2 ,,

This should be applied daily over the whole of the inflamed and swollen surface. In addition to this external dressing, one tablespoonful of sulphur should be administered to each affected animal daily. In the course of a week the swelling will gradually subside" (Soga). It is, therefore, important to observe that as soon as a swelling appears at the seat of inoculation, and from the swelling there exudes a yellowish serous fluid similar to the virus introduced, do not hesitate, but amputate above the swelling at once, and see that the tail bleeds freely. The bleeding may be stopped by a ligature, but it is more satisfactory to use a hot iron—a docking iron is best—and sear the wound. This prevents septic poisoning and secondary swelling, which are apt to follow, more especially when there are a number of swollen tails in a herd. To prevent this, it is also advisable to remove every animal with a badly swollen tail from the herd.

Does inoculation introduce lung-sickness into a herd of healthy cattle? The large majority of experienced colonial farmers are fully convinced that the disease does occasionally appear in a clean herd of cattle after inoculation, when there is no other source of infection known.

Admitting that such infection does occur, the question arises, How is the disease introduced? Do inoculated cattle communicate the disease to healthy susceptible cattle with which they come into immediate contact? The opinions of colonial farmers are somewhat divided on this point, but the general opinion of European experts, arrived at by close observation and experience, is that healthy cattle inoculated in the tail in the usual manner do not communicate the infection to healthy susceptible cattle with which they mix, and the somewhat extensive experience of our own veterinary staff supports this view.

(NOTE.—There is, however, little doubt, from the observations

of Hutcheon and Gray, that in highly susceptible stock lung lesions may develop occasionally in such animals after inoculation, as cases of this sort have been observed in inoculated animals brought from areas in which the disease has never existed.)

The After-Treatment of Inoculated Cattle.—Close attention should be given to inoculated cattle from the fifth day onwards. After this the cattle should be regularly kraaled, and particular attention should be given to the animals when they rise in the morning to see if they manifest any stiffness or inability to lift their tails. Any such symptom should lead to the animal being caught and its tail examined at once.

By the tenth day the inoculation should have taken in a considerable portion of the cattle. This taking is manifested by a slight swelling at the seat of inoculation, with a beady exudation from the surface of the skin.

Is it possible, then, to communicate lung-sickness to healthy cattle direct by inoculation at the tail? The large majority of European experts express a confident opinion that you cannot, and when the disease does appear in an apparently clean herd after inoculation, they assert that the infected animals must have contracted the infection previous to their inoculation, or before the inoculation has had sufficient time to confer immunity on them. Of course, it is impossible to prove the contrary, more especially as inoculation against lung-sickness is usually resorted to only when the disease has already appeared in the herd itself, or amongst some herds in the near neighbourhood.

But from the numerous instances in which I have observed the disease to follow inoculation, when no other reasonable explanation could be offered except that of direct communication by the inoculation, I am thoroughly convinced that inoculation is capable of communicating lung-sickness direct to a healthy susceptible animal. I am quite ready to admit that such cases are comparatively rare, but that they do occur and are the means of introducing the disease into a previously healthy herd I entertain no doubt whatever, and it does not appear so improbable that such cases should arise under special circumstances.

It is a well-established fact that if an ox which has been inoculated or drenched as a preventive of lung-sickness is operated on for any other cause, or receives any external injury during the time that the specific virus is circulating in the blood, specific local swellings are liable to occur at the seat of injury and often assume serious proportions, proving fatal in many cases. May not the same local manifestation of the disease occur in the lungs

through the accidental rupture of a small bloodvessel or local injury to the lung tissue, thus permitting the organisms of the disease to escape into the interlobular tissue?

Successful Inoculation.—It is necessary to bear in mind that inoculation does not render any animal immune unless it has taken properly.

There is another point of equal importance to remember, and that is that an inoculated animal, even when the operation is successful, is not rendered resistant to infection until after the inoculation has taken effect, which at the earliest is about the eighth day, and very often as late as the twenty-first. This shows that all the cattle in an inoculated herd are liable to become affected with lung-sickness for at least eight days after inoculation if exposed to infection, and many of the successfully inoculated ones for a much longer period, while those in which the inoculation has completely failed to react have no immunity conferred on them whatever; hence they are liable to contract the disease at any time if exposed to infection. This shows the necessity for carefully removing every affected animal from the herd in order to remove all sources of infection.

It is also necessary to mark all the cattle in the herd in which the inoculation has failed to take properly. These should be re-inoculated at the end of a month. Unless these precautions are taken, the disease may linger amongst the susceptible cattle in the herd for an indefinite time.

Drenching.—With respect to drenching—that is, the administration by the mouth of a certain quantity of the fluid found in the thorax—as a preventive of lung-sickness, the experiments conducted by the veterinary staff, in addition to the previous practical experience of hundreds of colonists, have clearly shown that drenching is an effective preventive against an attack of the contagious lung-sickness of cattle when properly carried out. It is also comparatively free from danger if proper precautions are taken and due care exercised in administering the fluid. The following are the points which should be carefully attended to:

The fluid found in the chest of an affected beast should alone be used for drenching, and it should be used pure after being strained; no water should be added to it, but the mouth of the beast may with advantage be washed out with clean water after the dose has been administered. This is merely to clean the mouth and prevent local inoculation, should there be any abrasion of the lining of the mouth. Some place a tablespoonful of common salt in the beast's mouth immediately after

administering the dose of lung-water to clean the mouth, and it is an excellent plan. I have used it largely myself.

Experiment has proved that large doses of the lung-water may be administered with safety, but I would recommend for full-grown beasts about 10 fluid ounces or a large breakfastcupful; less for younger animals in proportion to age.

What is the best method practicable of eradicating pleuro-pneumonia from our herds of cattle? There can be little doubt that the quickest and surest method of eradicating pleuro-pneumonia from our herds in the colony and in South Africa generally would be to slaughter, not alone the affected, but all the cattle that had been in contact with them. This is the method which was adopted in England and in every other country which has successfully stamped this disease out. But the pleuro-pneumonia of cattle is so widely prevalent amongst the cattle in our native territories, and there are various other reasons why such a measure could not be successfully carried out, that it need not be further considered.

In conclusion, I may remark with regard to the method of drenching, in addition to the difficulty or impossibility of obtaining a sufficient quantity of fluid from destroyed animals to drench a large herd of cattle, that the method was found to cause too great a number of fatalities to justify its continuance, and it was dropped. Of the other methods detailed, perhaps the syringe gave fewer cases of septic infection, with the inflammation extending from the seat of inoculation up the tail into the hind quarters, and death resulting from blood-poisoning; but, speaking from a rather extensive experience of inoculations that I have seen for this disease, I can only remember with a shudder the many poor mutilated cattle that have resulted.

Susceptibility to the Effects of Inoculation.—There is a great difference in the susceptibility of stock to the effects of inoculation. The susceptibility is least amongst stock coming from areas in which the disease has been enzootic for many years, and most marked in animals from an area where the disease is unknown; and when it becomes necessary to inoculate animals of the latter class, unless they are very carefully watched when the inoculation begins to take, heavy losses may follow.

If inoculation for the eradication of the disease is to be carried out in the future, the inoculating material should be scientifically prepared and supplied by a veterinary laboratory; but if it should be the misfortune of Rhodesia or other parts of South Africa that have been free of the disease for a number of years to import

lung-sickness, unless the circumstances are very exceptional, there is little doubt that the soundest and most economical method of eradicating the disease will be by slaughter of all sick animals and all animals that have been in contact with them.

I fully realise that this method of dealing with disease is distasteful to many South Africans, but where circumstances permit of it and justify its adoption, it is the only sure way of eradicating lung-sickness.

TUBERCULOSIS

Synonyms.—Consumption, Phthisis, Phthisis Pulmonalis, Scrofula, Pining, Tabes Mesenterica, Pearl Disease, Grapes, etc.

History.—Tuberculosis appears to have been recognised from the earliest times, and the contagious nature of the lung form of the disease was apparently suspected by physicians of antiquity, whilst provision against the consumption of tuberculous flesh was set out in the Mosaic laws. In 1865 Villemin confirmed the suspicion of the contagious nature of the disease by the experimental inoculation of human tubercular material into animals. In 1868 extensive feeding experiments were carried out by a number of different investigators which demonstrated the transmissibility of tuberculosis by ingestion.

In 1882 Koch discovered the causal bacillus of the disease—the *Bacillus tuberculosis*, Koch's bacillus, or the tubercle bacillus, as it is variously called—and he considered the bacillus to be identical in man, cattle, and birds. In 1884 he reaffirmed his conclusions of two years previously, after having found the bacillus in a greater number of species of animals; but in 1901 Koch startled the scientific world by retracting his earlier opinion and stating that human and bovine tuberculosis were distinct affections, and consequently the possible infection of human beings from the ingestion of meat and milk from tuberculous animals was remote, and that special preventive measures in this direction were not necessary.

Koch's theory of 1901 was not upheld by many authorities, and at the present time the transmissibility of the disease from the lower animals to man is firmly established and recognised.

Tuberculosis is found in most parts of the world, but is said to be rarely met with in the Arctic Circle; it is possible that the low temperature of that region exerts a deleterious effect upon the organism outside the body of its host, and may to a great extent hinder the spread of the disease by inhalation and other ways.

Bovine tuberculosis has probably been present to a limited extent in South Africa for a number of years. The earlier reports of the Veterinary Division of Cape Colony indicate that the

disease was practically non-existent, except in Cape Town and its vicinity, and it was of opinion that if it could eradicate the disease by means of the tuberculin test in that locality and prevent its reintroduction by cattle imported from overseas, it would be possible to keep South Africa free of the pest. Present-day experience appears to indicate that this was a rather too sanguine view of its incidence in the country.

In 1902, after the Boer War, to replenish the cattle herds of the country, animals were imported from, amongst other places, Madagascar; these animals were badly infected with tuberculosis. Veterinary Surgeon R. Robinson reported that, of a shipload landed at East London, 30 per cent. reacted to the test and revealed the lesions of the disease on post-mortem examination; further, in others of these animals that died from redwater and from lung-sickness, in every full-grown animal tubercular lymphatic glands were found.

In 1908 measures for the eradication of the disease in the Cape were started. Thirty-eight outbreaks of the disease were reported in the districts of Albany, Cape, East London, Glen Grey, Malmesbury, Paarl, and Stellenbosch, affecting 1,335 cattle; of these, 181 died of the disease or were destroyed. At that time cattle in those localities, more than anywhere else in South Africa, were housed—that is, kept under conditions that favour the spread of the disease. In course of time, as other places, notably Johannesburg, commenced to house cows, outbreaks of the disease occurred. Rhodesian experience apparently indicates that there is just sufficient disease present in the indigenous stock of the country to lay the foundations for the disease to take a firmer hold if and when cattle are housed together as in dairies.

Rhodesian experience of tuberculosis is somewhat unique and interesting. The disease, so far as I am aware, has never been clinically diagnosed (ante-mortem) in Rhodesia, and prior to 1916 it was believed not to exist in the country. In that year an export trade in slaughter cattle with Johannesburg commenced, and from then to the present day approximately 60,000 animals have been slaughtered at that town, of which thirty-two have been found to be affected with tuberculosis. The infected animals have not been found amongst the limited number of grade cattle that have been exported, but amongst the unimproved native cattle. Neither have the infected animals come from one herd or even from one locality or district; they have been drawn from practically all parts of Matabeleland. It is difficult to account for the way in which these animals have contracted the disease.

Most, if not all, of them have never seen the inside of a building. Of course, a number of the native owners of such cattle are tuberculous, which fact suggests one possible source of infection.

Nature of the Disease.—Tuberculosis may be justly described as the most widespread of all diseases amongst animals, but it is unevenly distributed in the various species. In the domesticated animals the disease is most commonly met in cattle. It is difficult to say if the disease is more common in man or in cattle. In the matter of frequency, next to cattle comes the pig or the fowl. In the horse and the domestic carnivora the disease is not common, nor can it be called rare. In the sheep it is distinctly rare.

The frequency with which a species of animal is affected with disease is not always a correct measure for its susceptibility—for instance, sheep are readily infected with tuberculosis experimentally, and their freedom from this disease under natural conditions is due to the fact that their outdoor life minimises the risk of infection. The same rule applies to cattle, housed cattle being far more commonly affected than those running at large, simply because their indoor existence greatly multiplies the chances of infection.

Goats, like sheep, under natural conditions are rarely affected with the disease, but where they are stabled for milking purposes they readily contract the disease and become affected to the same extent as cattle.

The rabbit and the guinea-pig, especially the latter, are remarkably susceptible to bovine tuberculosis by experimental inoculation, but spontaneous tuberculosis naturally contracted amongst wild rabbits is unknown, and in rabbits and guinea-pigs confined the disease has little tendency to spread. The mouse and the rat are not very susceptible to the disease, but they can be experimentally infected.

Wild animals in a state of nature are rarely found to be affected, but amongst animals kept in confinement, such as in zoos and menageries, the disease is not rare. Monkeys are very susceptible.

Methods of Transmission of the Disease.—These are inhalation, ingestion, inoculation, intramammary and intravaginal infection; probably all these methods are occasionally or frequently in operation naturally, and to them may be added intra-uterine infection.

Relative Frequency of Methods.—Until a few years back it was all but universally admitted that the common method of the spread of the infection was by inhalation of the dry bacilli floating

in the atmosphere. This was held to be a legitimate conclusion from the fact that the lungs or their lymphatics are the commonest seats of the disease, and that they are apparently the primary seats of it.

If 1,000 slaughter-house cases of the disease are taken, and the apparently primary seat of the disease, and also cases in which only one organ is infected are tabulated, the lungs will be found to be the organ in about 90 per cent. of the cases.

The primary theory of inhalation as the common method of infection was followed by the ingestion method being put forward as the common source of spreading the disease, and the occurrence of the majority of the lesions being in the lungs was accounted for by supposing the lung tissue to constitute a predilection seat for the disease, similar to the buccal mucous membrane being a predilection seat in foot and mouth disease.

It may be held that the common primary seat of tubercular lesions does not give a clue to the method of infection, because man or cattle infected by ingestion may have primary lesions in the lungs. Assuming that the tubercle bacillus is absorbed from the intestines into the lacteals, it passes next through the glands into the thoracic duct of the blood-stream, and thence to the lungs.

It would appear that in some animals—goats as an example—primary thoracic lesions have been experimentally obtained after ingestion, no abdominal lesions being present. It is not possible, or very rarely so, to get that result when other animals, particularly cattle, are infected by ingestion. In such cases there are mostly abdominal lesions present, especially in the mesenteric glands; often the lesions are exclusively abdominal.

In conclusion, it may be held that the evidence that ingestion is the common method is incomplete, but it has always been admitted that a considerable number of cattle are infected by ingestion, and that it is the commonest method in calves.

Infection by Inoculation.—In cattle this method of transmission in nature is almost unknown, but cases of skin tuberculosis are sometimes met with.

Direct Infection through Teat Canal.—This is a rare method, but there are on record what appear to be cases of primary tubercular mammitis.

Intravaginal Infection.—This is also a rare method, but again there appear to be cases of primary tubercular inflammation of the womb. The infection could be caused by an infected bull with diseased genital organs or by a healthy penis soiled with tubercle bacilli from a previous cow.

Congenital or Intra-uterine Infection.—This was supposed to be a common method of infection in both human beings and in cattle, but it is now admitted only quite a small percentage of cases arise in this way. In order to prove the origin of the disease during intra-uterine life, the actual lesions of the disease must be shown at birth or very shortly afterwards. To find the disease in an animal a month old is not proof that the disease was contracted in the uterus, because young animals may contract tuberculosis and die within a month.

The belief that bovine tuberculosis was congenital was in the first place disproved by slaughter-house statistics. Further investigation revealed the fact that in herds where from 20 to 40 per cent. of the cows in them were infected with tuberculosis, the proportion of calves suffering from the disease under a month old was very small—certainly less than 1 per cent. In badly infected herds one calf in two or three undred is born suffering from tuberculosis.

Intra-uterine infection appears to be very much less in other species of animals, and it is very rare, if ever, it occurs in mankind. The relatively larger number of cases in bovines is due to the more frequent existence of tuberculosis in the uterus of cows. When cases occur, they may be due to one of the following:

Placental Tuberculosis.—When the disease is present in the womb, the tubercle bacilli are carried with the blood to the fœtus direct, and the primary lesions of the disease are found in the liver.

Conceptional Tuberculosis.—The ovum at the act of conception or soon after becomes infected, although the uterus is healthy. If the male parent had to do with the transmission of the disease, this would be the way it would happen. It is possible there are cases brought about in this way, but it is not certain. If they do occur, they are exceedingly rare, because testicular tuberculosis is rare; and, further, if tubercle bacilli reached an ovum at the time of fertilisation, it is scarcely conceivable that the ovum would not be immediately destroyed.

Infection in the Pig.—In the immense majority of cases the lesions of the disease indicate quite clearly that the infection has been caused by ingestion. The same distribution of the lesions are also obtained in pigs experimentally fed with infected materials.

Throat lesions are present in many cases in the tonsils, but more frequently in the pharyngeal lymphatic glands; failing or in company with the preceding, abdominal lesions are found.

It is possible some cases occur from inhalation. The congenital method appears to be rarer than even it is in cattle.

Pigs are frequently the means—through being fed on skim milk and their coprophagous habits—of finding out the existence of tuberculosis amongst the cattle stock on a farm.

Infection in the Horse.—With few exceptions, in all the recorded cases of the disease in the horse the lesions have indicated infection by ingestion; in perhaps 99 per cent. of the cases this method has been indicated. In the exceptional cases the lesions have been in the thorax, and were probably due to inhalation.

Infection in the Dog and Cat.—The lesions are frequently abdominal, some are thoracic.

Infection in the Fowl.—Ingestion is apparently almost the invariable method of infection.

The Causal Organism.—The tubercle bacillus is an obligatory parasite in all parts of the world; there is no evidence whatever pointing to tuberculosis ever being sporadic. It is very likely that in the remote past the tubercle bacillus had a saprophytic ancestor, possibly an acid-fast, saprophytic, non-pathogenic organism.

The main reason against the tubercle bacillus being saprophytic is that it requires a high and constant temperature for its growth. It must have a temperature not below 90° F., but it grows best at the body temperature.

In the dead animal the organisms are found to be, broadly speaking, confined to the lesions or the discharges from them. Even in the most susceptible species of animals, such as the guinea-pig and the rabbit, the disease is hardly ever septicæmic. In the larger animals the bacilli are never found in the blood by microscopical examination. There is no reason to believe that the bacilli propagate in the blood in any animal. It is a tissue parasite; it is transported by the blood-stream, but it does not multiply therein, and it does not make the round of the circulation. The bacilli soon become arrested in the capillaries, mostly in those of the lungs.

Dissemination of the Organism in the Body.—When tubercle bacilli have established themselves in any part of the body they may spread—

(*a*) By direct extension, the same as a cultivated colony of the organism extends on a plate. This method is unimportant.

(*b*) By the lymphatic vessels. This is one of the most important ways of the spreading of the disease within the body. From a primary focus the tubercle bacilli are carried with the flow of lymph,

but they are more often transported with the leucocytes which have ingested them; they are thus soon within the lymph vessels, and are rapidly transported to the lymphatic glands. This explains the fact that if the disease is of any standing in an organ, the contiguous lymphatic glands are always diseased. Pulmonary tuberculosis with healthy mediastinal lymphatic glands is exceedingly rare.

In the majority of cases the bacilli spread the same way the lymph stream flows—that is, from the periphery to the centre of the body; sometimes, though, they spread in an opposite direction, as, for example, if an animal is inoculated in the middle of the neck, the pharyngeal glands as well as the prescapular may become infected. Similarly in natural cases the disease spreads from the peritoneum to the precrural glands, also to the supramammary glands and the udder.

(*c*) Along the free surface of the mucous and serous membranes of the body. Once a focus of the disease has established itself in the lung, the bacilli escape to a bronchus, and then the respiratory movements and acts of coughing spread them still more widely, and in this way new parts of the same or the other lung may be infected. In all bad cases of pulmonary tuberculosis some bacilli are coughed up and swallowed again, thus giving rise to secondary lesions in the pharyngeal glands or in the abdomen.

When once a spot of the pleura becomes tuberculous, the disease overruns the greater part in a short time, partly by the rich lymphatic network in the pleura and partly by the respiratory movements. Precisely the same thing happens in the case of the peritoneum. Infection of one of these membranes, pleura, or peritoneum may lead to the infection of the other by the lymphatic vessels passing through the diaphragm.

(*d*) By the blood-stream. In natural cases this appears to be quite an exceptional occurrence; when it does occur, acute generalised tuberculosis results. When the disease is not spread by the blood-stream it is a localised tuberculosis, even if it spreads over a large area. In cattle and in human beings, when the disease is allowed to run to a fatal termination, generalisation of the disease by the blood-stream may not occur. It is not often that it does so.

The Manner by which Tubercle Bacilli get into the Blood-Stream.—1. When the disease involves the wall of a vein, the lining membrane (endothelium) gives way, and the bacilli obtain entrance into the lumen of the vessel; but this method is not of common occurrence.

2. The most frequent way is for the tubercle bacilli to escape from a diseased and disorganised lymphatic gland into the thoracic duct. In such a case the bacilli first reach the capillaries in the lung; the majority are there arrested and set up acute miliary tuberculosis, which is the only reliable naked-eye mark of generalisation of the disease by the blood-circulation. In such cases all the spongy lung tissue is filled with these tubercles, that are only visible to the naked eye about a fortnight after generalisation has occurred. This condition is rapidly fatal. Some of the bacilli generally pass through the pulmonary vessels into the left side of the heart, and are from there distributed throughout the whole body to every organ and tissue according to the amount of their blood-supply. The subsequent development of the tubercle is then very unequal; muscle and tendinous tissue escape, more or few evolve in the liver and spleen, less evolve in the kidneys.

Even when tubercle bacilli are injected direct into the veins, few or none may evolve in the kidney and muscles; the only explanation for this must be that either the tubercle bacilli are not present everywhere in the capillaries, or that special organs and tissues are not favourable for their development.

A remarkable fact is that in a great majority of natural cases in cattle (and man) the disease tends to run a very chronic course. In the majority of subjects no visible symptoms of ill-health are present, and the disease does not cause death. A cow may suffer for years and appear to be quite well, and when killed the disease may be found to be of very small extent—perhaps confined to a few mesenteric glands.

This is in marked contrast to the experimentally produced disease. If a young ox is inoculated, an acute generalised tuberculosis results that is fatal within two months. In such a case there is always a formidable lesion at the seat of inoculation, as well as a host of miliary tubercles in the lungs, liver, spleen, and in nearly all of the lymphatic glands and bone-marrow. The explanation may probably be that it is difficult to experimentally imitate natural infection with a very small number of bacilli, which enables the animal to evolve a certain degree of immunity and so hold the disease in check. When inoculated experimentally its defences are, in a manner of speaking, rushed.

A hitherto unknown fact was shown a few years back by the British Royal Commission on Tuberculosis, and that is that when a moderate dose of tubercle bacilli of comparatively slight virulence, such as many human strains are, is injected under the skin

of an ox, there is a dissemination of these bacilli throughout the body. This may be missed unless the animal is killed and the tissues are searched microscopically; otherwise the animal may develop no symptoms, and when killed show no lesions at all.

Variations in Virulence of Tubercle Bacilli.—The bacilli which are found in tubercular lesions vary only within narrow limits of virulence, morphological or cultural characters.

Bovine bacilli are intensively pathogenic for bovine animals, also for the guinea-pig, rabbit, and a great number of other species.

Their chief cultural character is that they grow in a meagre sort of way, especially in primary cultures; therefore they are called " dysgonic cultures " (or growing with difficulty).

Human Tubercle Bacilli.—The cultures of tubercle bacilli from a large series of human cases are found to fall into two different groups in respect of virulence and cultural characteristics.

One group contains bacilli indistinguishable in any character from bovine bacilli. This group, in the great majority of instances, is found in cases in which disease is either confined to the abdomen or in which the primary lesions were obviously abdominal. Such cases have been found principally in children or young persons.

Considering that a large proportion of primary abdominal tuberculous cases in human beings are caused by bacilli belonging to this group, it is only fair to draw the legitimate inference that the infecting bacilli have come from cattle, probably by way of the milk.

The other group of bacilli includes strains which are for certain species of animals less virulent than bovine bacilli, and that grow more freely as cultures, and are therefore called " engonic cultures "; but this character is not of great value for separating the two classes, because the least dysgonic bovine bacilli merge into the least engonic human bacilli. The difference in virulence is much sharper. The bacilli of this group possess only a feeble virulence for cattle. Even when calves are inoculated with very large doses a fatal infection is rarely set up. There is often a considerable local lesion, with the neighbouring lymphatic glands affected, and if the animal is killed scattered tubercles may be found in various organs, but if the animal is allowed to live it usually recovers and the lesions disappear. Another marked difference is that the bacilli are not fatal to the rabbit. The

guinea-pig is readily infected with the human strains, but survives longer.

The question whether human or bovine bacilli are more fatal to man can hardly be definitely settled, as experimental inoculation of man is impossible, but it has been found that bovine bacilli are as virulent as human bacilli for man's nearest relative, the chimpanzee.

Bacilli in Pigs.—In a great majority of cases the bacilli are of the bovine type; there is no reason to doubt they are actually bovine bacilli conveyed by the milk and by the fæces of tubercular cattle consumed by pigs. In a minority of cases of pig tuberculosis the lesions contain avian bacilli, and in a small number of cases human bacilli.

Bacilli in Horses.—The bacilli are sometimes of the bovine type. It is said that avian bacilli have been isolated from equine lesions, also strains that are difficult to assign to any particular type.

Bacilli in Dogs.—Both types of bovine and human bacilli are met with.

Bacilli in Birds.—So far as is known, only one type of bacillus is found in farmyard birds, and this is known as the avian bacillus. It is decidedly engonic, and the cultures are unctuous or fatty in appearance, and not scaly.

The bacilli have hardly any virulence for the larger farm animals, but may in a natural way affect the pig. They have a very feeble virulence for guinea-pigs, but are pathogenic for the rabbit.

The disease is most frequently met with in the common fowl, but is also found in ducks and turkeys. It is very common in pheasants reared in confinement in Great Britain.

In the parrot bacilli of avian type are found, but sometimes the bacilli are distinctly of the human type.

Lesions in Different Species of Animals.

Cattle.—If one takes the statistics compiled of lesions in healthy —that is, killed for meat—slaughter-house cattle, the commonest seat of the lesion is found to be the bronchial and mediastinal lymphatic glands, and secondly comes the lungs. In a large proportion of cases the thoracic lymphatic glands are diseased, while the lungs are healthy; but it is very rare that the lungs are found diseased and the thoracic lymphatics healthy. It is assumed that the tubercle bacilli pass through the lungs by the lymphatics—without exciting lesions on their way—to

the thoracic glands, just as in quite a similar way they pass through the intestinal wall without provoking lesions until they reach the mesenteric glands, and in the case of the pharyngeal glands it is often the same.

In other cases where the disease is of small extent, only one or more pharyngeal or mesenteric glands may be diseased.

These four sets of glands are the commonest seats of the disease.

If an animal is allowed to die or is only slaughtered when it is near to death from tuberculosis, then the lesions are invariably extensive, and they practically always involve organs in both the cavities of the body. A common distribution is: Both lungs are the seat of a tubercular nodular broncho-pneumonia extensively caseated, with the bronchial mucous membrane ulcerated in places, and the bronchial and mediastinal lymphatic glands greatly enlarged and practically destroyed by caseation. These glands are, roughly, about the size of two fingers in a healthy ox, and the post-mediastinal and bronchial glands about the size of a walnut; in tuberculosis they may be as large as the fist. Pleural tuberculosis is also present. This is never primary in natural cases; it is usually secondary to tuberculosis in one or both lungs. It is sometimes secondary to tuberculosis existing in the thoracic lymphatic glands. These lesions are of a productive type, and on the pleural surface there follows a coarse warty growth known as grapes. There is a marked tendency in bovine tuberculosis to involve the serous membranes lining the great body cavities, and the peritoneum may also be found to be affected.

In a good number of cases the disease exists in the pharyngeal lymphatics, where it may have been primary, but it is probably more often due to auto-infection.

In the abdominal cavity it is common to find intestinal ulcers; these are usually confined to the small intestine. When they are fully formed they are crater-like in appearance. A few or more of the mesenteric glands are constant seats of the disease, and the lumbar and iliac lymphatic glands may also be affected.

Tuberculous nodules are frequently present in the liver and in the hepatic lymphatics; they are less frequently so in the kidney. The peritoneum is frequently involved in the same way as the pleura.

A remarkable feature is that in adult subjects the splenic substance is rarely involved, but in the acute form of the disease in young calves it is of very common occurrence. It is constant in acute tuberculosis caused by inoculation.

In a considerable proportion of cases in cows the uterus and the

mammary gland are affected. The uterus as a rule is of secondary infection from the peritoneum, but there may occur a rare case of primary tubercular mastitis (inflammation of the udder).

The lesions are distinctly rarer that affect the pericardium (heart sac) and the heart wall, the testicle, and the bones. The bones that are most frequently affected are the vertebræ of the dorsal or lumbar regions. The infection in these cases appears to be a centrifugal one, from previous diseased lymphatics in the region; the pericardium is infected from the pleura, and in adults the myocardium by way of the extension of the disease from the epicardium. In the calf this may not be the case.

In very advanced cases of tuberculosis in cattle some of the peripheral lymphatic glands may become diseased in this way, such as the axillary, the prescapular, the precrural; and even the popliteal may be diseased. A great deal of discussion has taken place as to how the glands become diseased. Some put it forward as a proof that the bacilli are disseminated by the blood-stream, but this view appears to be contra-indicated, because the lesions are sometimes unilateral or do not involve all of the lymphatic glands. The other view is that the disease has reached the glands by the centrifugal way of the lymphatics, the precrural from the peritoneum, and the prescapular from the thorax, or they may have been infected from the pharyngeal glands. The prepectoral (between the first ribs) and the suprasternal glands are often infected.

Mammitis.—The frequency of this lesion in the cow is an outstanding feature of bovine tuberculosis. For description see Mammitis (Tubercular), also under Diagnosis.

Congenital Lesions.—The bacilli in the ordinary way get into the calf's body via the umbilical veins, and as a result the liver is the most constant seat of the disease, because a great many of the bacilli are arrested in that organ. Those that escape reach the right side of the heart by the blood-stream; the bulk of these pass through the foramen ovale to the left side of the heart, and, being thus short-circuited, few lesions are found in the lungs.

From the left side of the heart the bacilli are then distributed throughout the body, and a few tubercular nodules may be found in the heart wall, spleen, kidneys, or lymphatic glands. In some cases the disease is quite extensive in the mesenteric glands; this is as a rule probably part of the general infection of lymphatics by way of the blood-stream.

Horse.—There is very little variety in the distribution of the lesions in this animal. The primary lesions are almost always

found in the abdominal cavity. Cases may be met with in which lesions that are apparently simultaneous with those of the abdomen are to be found in the pharyngeal or submaxillary lymphatic glands.

The mesenteric glands are constantly diseased. Next in order of frequency come splenic lesions; these are present in about 90 per cent. of cases. In a considerable number of cases are intestinal ulcers; when present, they are usually of great size, are mostly found in the small intestine, but are sometimes present in the large. It is rare to find any large lesions in the liver, but sometimes there are minute or microscopical tubercles present.

Due to the immense number of bacilli that travel from the mesenteric glands via the portal duct to the liver, cirrhosis of the liver of fine monolobular character is often present. The kidneys are practically never the seat of tubercle, but in many cases both kidneys are greatly enlarged, due to chronic venous congestion. In the great majority of cases no peritoneal lesions are present, but a " grapy " condition may be present on the diaphragm.

The enlargement of the mesenteric glands is frequently enormous, and cases have occurred where the glands weighed as much as 56 pounds. The tuberculous tissue is frequently the seat of a firm whitish caseation; it is only rarely that the glands calcify.

The splenic lesions are large tumour-like growths, the tissue is not much degenerated, and the lesions are easily mistaken for neoplasms (tumours). They are firm, yellowish-white in colour, and on section in a proportion of cases both the mesenteric glands and the splenic growths are found to be the seat of a peculiar softening into a caseo-purulent material. When this has occurred, the growths are always enormously rich in tubercle bacilli.

The abdominal lesions may be in existence for a long time without the animal showing signs of ill-health except emaciation. In many cases, if the animal is allowed to live on, the respiration becomes hurried, and when the animal has died or been killed a miliary tuberculosis of the lungs, with the tubercles either discrete or confluent, is found to have occurred.

The bones of the neck are also a common seat of the disease; it occurs in about 10 per cent. of the cases, and when present is a valuable aid to diagnosis. The animal has a stiffness of the neck, is unable to bend it or feed off the ground. After death, if the bones are examined by boiling them or by allowing putrefaction to remove the tissues, all the cervical vertebræ will be found markedly irregular on their surface, as the result of a chronic

tubercular periostitis. Sometimes the bodies of the bones and the joints are also involved.

The disease does not affect the other bones of the body, only those of the neck. No explanation can be given why this form of the disease should occur in the horse.

The Pig.—The distribution of the lesions in this animal closely resemble that of the horse, probably due to the fact that in the majority of cases the source of infection is by ingestion.

The lesions in the pharyngeal glands are commoner than in the horse, and are probably the primary seat of the disease in the great majority of cases; next comes abdominal lesions, and of these mesenteric lesions are the most common. Splenic lesions are frequent. Intestinal ulceration occurs in a number of cases. The liver is more frequently the seat of lesions than in the horse. Kidney and peritoneal lesions are rare.

There is a great tendency to generalisation of the disease in pigs, which results in miliary tuberculosis in the lungs and in most of the lymphatics throughout the body.

Lesions in the dorso-lumbar vertebræ are comparatively common.

Caseation and calcification of the lesions are more common than in horses, the caseous material being white in colour.

The Dog and Cat.—In these animals sometimes the lesions are primarily abdominal; sometimes they are primarily thoracic. The mesenteric glands are the most constantly affected part, but the enlargement is not great. The liver is very constantly the seat of tumour-like nodules up to the size of a filbert nut. These have a fleshy or sarcomatous consistence; there is often no caseation or calcification present, and these nodules are often mistaken for tumours. The disease later on often develops into a miliary tuberculosis, which principally affects the lungs.

The Fowl.—These animals are almost invariably infected by ingestion of fæces containing the bacilli through contamination of the food or otherwise, and the primary lesions are usually found in the intestinal walls, which rapidly take the form of ulcers. At the seat of these lesions the bowel wall becomes pouched, and the lesions are quite manifest in the unopened bowel. In fatal cases the spleen and liver are constantly the seat of numerous tubercles about the size of a kaffir-corn grain. All these lesions are liable to early caseation, also calcification; this is especially so with the bowel lesions.

Pulmonary lesions are exceedingly rare.

Diagnosis of the Disease during Life.

The methods of arriving at a diagnosis during life over and above the clinical symptoms presented are:

1. Employment of the tuberculin test.
2. Microscopical recognition of the tubercle bacillus.
3. Cultivation of the bacilli.
4. Experimental inoculation of the suspected material.

Tuberculin Test.—Before dealing with this test, and although the subject does not perhaps fall within the scope of a work of this description, it may be interesting, if only as an example of similar work, to explain the proceeding adopted in the manufacture of tuberculin.

Manufacture of Tuberculin.—This is brought about by cultivating the tubercle bacilli as a surface growth on glycerine broth. It is a matter of indifference whether the bacilli employed are of an ordinary human or an ordinary bovine type, though it has been said that when a virulent bovine strain is employed the reaction to the test occurs earlier. The human strain has the advantage that the growth in the broth is quicker and luxuriant.

The inoculated broth is incubated at the body temperature, and providing a good strain of the organism and good broth have been used, the surface ought to be covered with growth in a month to six weeks, and the maximum growth should be reached by the end of two months; when this has been attained the incubation is stopped. One must be sure the culture is a pure one, and to this end part of the surface growth may be examined microscopically; but as a rule this is not necessary, because the appearance of the growth is so distinct. If it is not pure, the difference in its appearance is at once noticed, and the flask is discarded; if the growth is impure, it often turns the liquid turbid.

When the purity of the flasks have been ascertained, they are placed in a steam steriliser and steamed for two hours. This is partly for killing the tubercle bacilli, but one minute would suffice for that; the other part of the object is to extract from the dead bodies of the bacilli the substances responsible for the reaction in the tuberculin. The glycerine broth is then ready to be filtered, but before doing this carbolic acid is added to it in the proportion of $\frac{1}{2}$ per cent. This is added to serve solely as an antiseptic, not as a germicide, and for the reason that during the subsequent manipulations accidental (aerial) bacteria are sure to drop into

it, and the tuberculin would rapidly spoil and putrefy; but the carbolic acid prevents the majority of bacteria from growing, except certain bacteria such as staphylococci, which will grow in ½ per cent. carbolic acid.

The next procedure is to filter through a Berkefeld filter (though filter-paper will do), and a liquid is obtained perfectly limpid and free from bacilli. An occasional bacillus in the liquid is of no importance, but if many are present the tuberculin, when injected, may set up a severe local infection.

Contrary to the opinion generally held, this tuberculin is very stable; even in tropical climates it remains unimpaired probably for years. It is not impaired by light, although the instructions issued are for it to be kept in a dark place. The following directions are suggested by Sir John MacFadyean for the employment of tuberculin:

1. While under the tuberculin test, cattle ought to be kept in the house, fed on their usual food, and protected from draughts. They ought not to be allowed to drink large quantities of cold water between the sixth and eighteenth hours after injection. It is well to take their temperature at least once on the day preceding the test.

2. The dose of tuberculin for a medium-sized cow is 3 c.c., or 50 minims, and it may be varied above or below that according to the size of the animal. Large bulls ought to receive 4 c.c.

3. It ought to be injected under the skin with a clean hypodermic syringe. The most convenient points are in front of the shoulder, or on the chest wall behind the point of the elbow. The best form of syringe is one with an asbestos piston, and the whole instrument may be sterilised by boiling it in water for five minutes before use.

4. The tuberculin must be injected into the subcutaneous connective tissue, and care must be taken that the whole dose is introduced.

5. Ordinarily the temperature must be taken at the time of injection, and at the ninth, twelfth, fifteenth, and eighteenth hours afterwards. When there is any reason to suppose that the animal may have been already tested with tuberculin during the preceding two or three weeks, it is advisable to take the temperature at the third and sixth hours as well as at the times just mentioned.

6. Animals in which the temperature during the eighteen hours following the injection rises gradually from the normal (between 101° and 102°) to 104° or more may be classed as tuberculous. When the maximum temperature attained is under 104°, but over 103°, the case must be considered doubtful, and the animal may be retested after a month.

7. The test is not reliable in the case of animals in the last stage of the disease, or in those in which the temperature is over 103° before injection.

8. The tuberculin should be kept in a cool place, and protected from light. Should it become turbid or cloudy it must not be used.

9. The tuberculin test does not render the milk in any way injurious.

These are a few important points that should be noted in the use of tuberculin. It is mostly employed subcutaneously.

A large experience indicates that somewhat more than 90 per cent. of tuberculous cattle will react when tested with tuberculin, provided they have not been previously tested within a short time; healthy animals or animals suffering from any other disease will not react to the test.

A non-tuberculous animal may have its temperature sent up from some other cause during the time it is undergoing the test, and so appear infected, if the character of the reaction is not considered as well as its intensity.

Tuberculin is frequently but erroneously classed with the toxins, but an immense quantity can be given to cattle or guinea-pigs without making the animals ill.

With regard to the 10 per cent. of tuberculous cattle that give no reaction when the test is applied, failure to respond to the first test shows that the animal is as a rule at a very advanced stage of the disease, emaciated, and with very extensive lesions; but tuberculin also, for some unknown cause, does fail in a few animals that are only slightly affected. There are also cases in which tuberculin appears to give a wrong indication—probably not more than one in two or three hundred.

Some animals will react several times in succession to tuberculin when the injections are repeated within only a few days' interval of each other; others will not react unless a month is allowed to elapse before retesting; while in other cases a very marked degree of tolerance follows inoculation, and subsequent tests may be unreliable and misleading.

The great majority of animals can have their sensitiveness to the test completely removed by repeated injections of tuberculin.

When an animal has been recently fraudulently tested, a reaction may sometimes be obtained by giving two or three times the ordinary dose and taking the temperature once every two hours. Such animals often react in two to six hours, the temperature coming down rapidly after that.

Other Methods of Applying Tuberculin Test.—In 1907 Von Pirquet discovered that if tuberculin is rubbed into a shaved and lightly scarified area of skin, a remarkable difference in appearance results if the person is suffering from tuberculosis or is healthy.

When this test is employed it is not the ordinary, but the crude tuberculin manufactured by Koch's method that is used, diluted with its own volume of sterile water.

In non-tubercular subjects the test has no more effect than if just plain water was rubbed in, and the scarifications rapidly heal up. In tuberculous subjects the part becomes the seat of quite a lively inflammation that persists for four or five days.

This test, which is known as the cutaneous test, has been applied in cattle, but its reliability does not compare favourably with the subcutaneous method.

Conjunctival Test.—This test was also discovered in 1907, but by Calmette, and has been applied to cattle. It consists of dropping a few drops of tuberculin free from glycerine and carbolic acid into one eye of a beast. In healthy animals no reaction follows, but in a tuberculous beast, usually within twenty-four hours, a swelling and reddening of the mucous membrane of the eye occurs, accompanied by a flow of tears, which is followed by a purulent discharge that collects in the inner corner of the eye and extends to the face. This reaction may commence to fade about the twenty-fourth hour, or it may persist longer up to three or four days.

The weight of opinion is perhaps in favour of this test not being so reliable as the subcutaneous one. Some practitioners favour applying the two tests in combination, the subcutaneous seven days after the conjunctival.

The ophthalmic test is employed where fraud is suspected, as a previous injection of tuberculin does not influence its reaction. It is also useful in cases where a high temperature persists.

The Ophthalmo-cutaneous Test.—Consists in first applying the ophthalmic test and a few days later testing by means of the subcutaneous method. This test is highly spoken of as frustrating fraud. When the subcutaneous injection of tuberculin is made, if the animal is tuberculous, a secondary reaction occurs in the eye.

The Epidermal Test.—Consists in rubbing a tuberculin ointment into the skin. This method is perhaps even more unreliable than the cutaneous one.

The Intradermic Test.—In this the tuberculin is injected into the substance of the skin at the side of the base of the tail. A swelling that persists for a day or two and about the size of a walnut occurs if the animal is tuberculous.

Microscopical Recognition of the Tubercle Bacillus.—The tubercle bacillus is what is known as an acid-fast bacterium, and for some time after its discovery it remained the only known organism of this nature. Then the leprosy bacillus was discovered, which is also an acid-fast organism; this was followed by the smegma bacillus, found in preputial secretions, and which must be remembered when examining urine for the tubercle bacillus.

At the present time quite a number of acid-fast bacteria are known, the most important of which from a diagnostic point of view are those of grass and manure, the timothy grass bacillus being a well-known one.

Acid-fast bacilli are also found in butter; they are in all probability those of grass or manure. All these bacteria are saprophytes, and so far as is known none of them excite disease in animals. Some of them actually multiply within the milk system. Their importance is their possible presence as accidental organisms in lesions or discharges. They are distinguished culturally from the tubercle bacillus by growing rapidly and luxuriantly at a temperature below 90° F.

Another pathogenic acid-fast bacterium of the domesticated animals, in addition to the tubercle bacillus, is the one responsible for Johne's disease.

Microscopical recognition of the tubercle bacillus can only be practised when some accessible lesion or an available discharge from a tuberculous organ is present. In the case of cattle superficial lesions are not common, but in a small proportion of cases there are enlarged submaxillary, parotideal, prescapular, or precrural lymphatic glands, and sometimes these are actually discharging. Even when the gland is not discharging it can be lanced or harpooned for material for microscopical examination.

In human practice this method plays an immense rôle, especially in the case of sputum, but is only restricted in veterinary practice, as growths or expectorate, even in advanced stages of the disease, are seldom available in cattle; but it has been shown that by obtaining expectorate on swabs from the fauces (back of mouth) tubercle bacilli may be found. There is a much greater chance of error in diagnosis than in the human being, on account of the acid-fast grass bacilli that may be found in pharyngeal

mucus, so that one cannot pronounce, if only a few bacilli are found, that they are tubercle bacilli.

Riddock, a Glasgow veterinarian, found that when the wall or woodwork in front of a tuberculous cow was examined, mucus or expectorate on its surface might be detected which contained tubercle bacilli, and that these were not grass bacilli was evident because they were in considerable numbers and were associated with recognisable cells.

Fæces may be examined for intestinal tuberculosis, but such a method is valueless in herbivora on account of the acid-fast saprophytes present; besides, in countries where the disease exists one cannot distinguish between tubercle bacilli and the bacilli of Johne's disease.

To examine urine for the bacilli it must be first centrifuged, and smears made from the deposit obtained.

In cases of tuberculous inflammation of the womb the vaginal discharge usually contains tubercle bacilli.

Cultivation of the Bacilli.—In ordinary practice it is impossible to make any use of this method.

Experimental Inoculation.—This is the most delicate test of all. and any sort of available material such as a discharge or a scraping from an incised lymphatic gland can be employed.

The most convenient animal to use is the guinea-pig, and the material is injected either subcutaneously or intraperitoneally. If it is probable that there are many accidental bacteria present in the material, such as is usually the case in expectorate, the subcutaneous method should be adopted.

Diagnosis of Tubercular Mammitis.—From a public health point of view it is most important that tuberculous milk should not be consumed, and that tubercular affections of the udder should be early recognised.

Diagnosis of tubercular mammitis may be sometimes based, without much chance of error, in the gross character of the lesion in the udder. When it is of large extent and has led to a great increase in volume of one or more quarters of the gland, and where the history of the case is that the disease has been of a chronic character from the outset, and where the diseased tissue is markedly indurated, there is very little doubt that the case is one of tubercular mammitis.

There are many other kinds of mammitis (see Mammitis) in the cow, but the majority are acute; the disease develops within a few days, and leads to marked swelling that is hot and tender; the milk is immediately altered in appearance and the quantity

is reduced. If the cow survives the attack, the affected quarter shrinks and becomes smaller than it ought to be. There is hardly any other condition (see Actinomycotic Mammitis) except tuberculosis that leads to a hard, steady enlargement of the udder.

In every case of suspected tubercular mammitis it is advisable to search the milk microscopically for the bacilli. The richness of milk in bacilli is very variable; as a rule it is difficult to detect them at the outset of the disease while the milk is unaltered in appearance, but when the milk has altered in appearance and is sedimentary, there is little or no difficulty in finding the bacilli. Sometimes the bacilli are quite numerous. It is a waste of time to examine milk microscopically without first centrifuging it. After it is centrifuged a smear is made of the deposit and examined. If there is not much deposit present, it is advisable to examine the cream, as the bacilli may have been carried up with the cream.

When tubercle bacilli are suspended in a watery liquid or milk serum the whole of the bacilli tend to sink to the bottom, because they are heavier than the water; but when the milk is still rich the ascending fat globules carry with them a great number of tubercle bacilli.

To obtain milk for examination the teat must not be wetted, and the milk must not come in contact with the milker's hands, but must come direct from the teat into a sterile bottle. If the affected quarter is milked in this way, there is scarcely any chance of error in taking any acid-fast bacilli found upon examination to be tubercle bacilli.

There is no known acid-fast organism except tubercle bacilli capable of causing inflammation of the udder, but in rare cases acid-fast bacilli other than tubercle are found in the udder which are sometimes morphologically indistinguishable.

To test milk by experimental inoculation is a most delicate test that will often lead to a positive diagnosis where microscopical examination fails. As soon as a lesion has obtained microscopical dimensions, tubercle bacilli are passed out with the milk, and can be demonstrated by inoculation. The milk is obtained as for examination, and strict precautions are taken to prevent accidental contamination; it is centrifuged and 2 or 3 c.c. are injected into the peritoneal cavity of a guinea-pig, four of these animals being used for each experiment.

Sometimes milk not direct from the cow, such as shop or market milk, has to be examined for the tubercle bacillus. The procedure is the same as with milk direct from the cow, only some of

the rabbits must be inoculated subcutaneously on account of the accidental bacteria present in such milk.

A sample of shop milk or any other not taken with proper precaution, and that provokes tuberculosis, is not proof that the cow's udder from which the milk was taken is tuberculous, because all milk contains cow's fæces, and this is sure in some cases to contain tubercle bacilli, or the bacilli may come from the lungs. Tuberculosis of the womb, with a vaginal discharge, is another source of infection.

An important question is whether tubercle bacilli may be passed out with the milk from an udder that is not the seat of tubercular mammitis. This question must be answered in the affirmative. When this occurs the animal is usually in an advanced stage of the disease. When the tubercular lesions are only restricted and the udder is healthy tubercle bacilli are not passed out. These facts have been proved by numerous experiments.

When the tubercle bacilli are few in number in milk, some of the guinea-pigs will escape infection when inoculated with it.

Symptoms.—The disease is usually insidious in its development, and slow and chronic in its course, and, as pointed out under the history of the disease in Rhodesia and proved elsewhere, its presence may only be found out when fat cattle are slaughtered; therefore, it follows that no symptoms that can be regarded as absolutely diagnostic are shown in the early stages of the disease, but the observant cattleman with experience is often shrewdly suspicious of affected animals. A short, dry, intermittent cough easily induced is a very suspicious symptom. As the disease advances the symptoms become more pronounced, and the animals (cattle) are known as "piners" or "wasters," owing to their inability to thrive and their "hide-bound" condition. In far advanced cases, or in those in which the superficial lymphatic glands or the udder are involved, or where the lungs are seriously affected, the clinical diagnosis of the disease can be undertaken with a greater amount of certainty, but the symptoms presented will depend upon the organs or tissues affected.

Post-mortem Appearances.—To a person with some experience the lesions in cattle, pigs, and horses are so characteristic that a diagnosis may be based on them with practically no chance of error.

The extensive lesions in cattle, their distribution, caseation, and calcification, could hardly be mistaken for anything else, but

doubt sometimes arises where lesions are small and not caseated. In such cases diagnosis is obtained by microscopical examination of smears or sections of the lesions, or by experimental inoculation of guinea-pigs. In making such examinations, the possible presence of acid-fast saprophytes must not be forgotten.

The tubercle is very small at the commencement of its growth, and increases to the size of a pin's head or larger. A number of these small tubercles may coalesce and form masses of various sizes. When first visible to the naked eye, the tubercle is a small, translucent, homogeneous-looking thing; a little later it becomes opaque, denoting that central necrosis has started; later still it becomes yellow, denoting that caseation has commenced.

In bovine lesions caseation is a practically constant alteration; it is always preceded by, and follows quickly on the heels of, necrosis. In the completely caseated parts of a tubercle in the ox it is distinctly yellowish in colour, and fairly firm in consistence. Sometimes—and especially is this so in thoracic lesions—the caseated tubercles undergo softening, and there results a custard-like material. This softening is due to an invasion of the part by pus-producing organisms—staphylococci as a rule.

Another common alteration of the tubercle is calcification. The tendency to this condition varies in the different species of animals; it is most pronounced in the ox and the pig, and least so in the carnivora. Calcification is invariably preceded by caseation, and commences towards the centre of the lesions. In this condition sometimes the tubercles become of almost stony hardness; in other cases they are only perceptibly gritty; and in still other cases mortar-like. Various opinions as to the cause of this tendency to calcification have been put forward, but are unsatisfactory, and the cause is unexplainable. The salts of calcification (carbonate and phosphate of lime) are identical with, and in similar proportion to, those of the bones of the animal's body.

The presence of calcification in lesions does not prove that they are of long standing, as it may set in in lesions of under two months old in the ox.

A fibroid transformation of the lesions is seen in miliary tubercles, but is very rare in cases of natural miliary tuberculosis in cattle, or in lesions of any great extent. When present it points to a process of retrogression, and only occurs when the tubercle bacilli have ceased to multiply at the spot, and are being overcome by the defensive powers of the body. There is little or no necrosis present, and in time the tubercle is replaced by fibrous tissues, and sometimes the tubercles disappear altogether.

Treatment.—Tuberculosis is an incurable disease, and once a definite diagnosis has been arrived at, treatment is out of the question. Not only will it prove of no avail, but there is the risk of the disease being conveyed to other animals and mankind.

Protective Inoculation.—Tuberculosis is no different to any other disease, in the sense that it does not kill all that are affected with it, and at least in cases of bovine tuberculosis (probably in cases of human beings also) a complete recovery may take place.

It can be proved by experiment that such recovered animals for a time offer exceptional resistance to infection, proving that some degree of immunity has been obtained. Tuberculous cattle often offer a greater resistance to experimental injection than healthy animals. The rule appears to be that an animal that has naturally contracted tuberculosis acquires a considerable degree of immunity.

In the majority of cases of naturally contracted disease the course is a slow one, whereas in inoculated cases it is rapidly progressive and fatal.

As soon as it was found that there were in nature strains of the bacillus not fatal by inoculation to cattle, it was suggested that such strains be used as vaccines.

Von Behring is generally credited with being the first to advocate vaccination in this way in 1902. He was under the impression it was necessary to submit the bacilli to a process of attenuation, and put upon the market such a vaccine called bovovaccine, composed of living tubercle bacilli of the human type. There is no reason to believe that any passage through animals is necessary to render that type of bacilli safe for cattle.

It was recommended that the vaccine should be used on quite young calves, and two separate intravenous injections were employed. In one or two months after the second injection the calves attained the maximum degree of immunity. In healthy calves the vaccination is devoid of danger, and has been practised on a large scale in Germany.

Experiments have shown that calves so vaccinated have acquired a strong degree of immunity, and can withstand what would otherwise be a fatal dose of virulent bovine bacilli; but the results are not quite constant: some animals do not acquire such a high degree of immunity.

Subsequent experiments and observations appear to show that the immunity is not very lasting. Such immunised animals, when tested after more than a year, offer no special resistance, and when placed in contaminated premises have contracted

tuberculosis. To keep up the immunity it is necessary to repeat the vaccination annually.

An unavoidable defect in the method is that it takes four or five months to acquire serviceable immunity, and this is just at that period of the animal's life when it is most liable to become naturally infected with the disease.

There is no evidence that the vaccination is at all curative.

The vaccination is not permissible in cows or heifers giving milk or about to calve, because bacilli may persist for months in the animal's body even when vaccination is done subcutaneously. The bacilli are distributed throughout the whole body, and while they would not be dangerous to the calf if present in the milk, they would be to human beings consuming it.

Prevention.—The prevention of tuberculosis in animals is a subject of paramount importance, both in the interest of the stockowner and for human health.

The methods that should be adopted for its control or eradication in South Africa deserve very serious consideration. The first point to be tackled apparently is to prevent the introduction of further cases of the disease into the country from overseas; for this the tuberculin test is employed. The next point appears to be to prevent the dissemination of the disease within the country from infected herds and localities to non-infected herds and localities; and, lastly, the eradication of the disease in infected herds.

The comparatively simple plan of testing with tuberculin and destroying the affected animals cannot be adopted in most countries, owing to the enormous cost it would involve; and the same objection to this method holds in South Africa also. Two other methods of controlling the disease have been adopted in Europe with considerable success; one was suggested by Bang in Denmark, the other by Ostertag in Germany.

Bang's Method.—This consists in testing the herd with tuberculin and then segregating the "reactors" from the healthy animals. The latter are put in tubercle-free buildings, and are not allowed to come in contact with any animal unless it has been subjected to the test. To avoid accidents and animals being missed by the test, it is customary to test the apparently healthy portion of the herd at regular intervals of six or twelve months.

The reacting cattle are kept in separate buildings and fields, and are bred from and milked for a time as usual, but are gradually fed off for the butcher. The calves are taken from their dams at birth and hand-reared, and are not allowed into the healthy

herd until they have successfully undergone two or more tuberculin tests.

It will be seen that this method does not disastrously affect the stockowner's business, and if efficiently carried out it ultimately results in a herd free from tuberculosis.

Ostertag's Method.—This method is to immediately slaughter all animals affected with "open" tuberculosis—*i.e.*, cases in which the disease occurs in those parts of the body having natural outlets, and thus being dangerously infective to other animals and to man. The trouble with this method is the determination of "open" tuberculosis, and when a non-infective animal becomes infective. This method was practically the one adopted by the British Board of Agriculture in 1909, but it was subsequently withdrawn.

The question of compensation for animals destroyed is the rock upon which most measures for controlling or eradicating the disease are wrecked. Broadly, Bang's method would appear, with our present knowledge of the disease, to be the one best adapted to meet South African conditions. I have given its principles very crudely, and it would need more elaboration—for instance, it would not be sound policy to keep alive animals that were disseminating the disease greatly even in a herd of reactors, neither would it be economic to attempt to fatten an animal for the butcher in which the lesions of the disease were so extensive that when the animal was slaughtered the greater part or the whole of the carcase would be condemned as unfit for human consumption. In connection with the latter point the Royal Commission on Tuberculosis in Great Britain, in its report in 1898, recommended that the following principles should be observed in the inspection of carcases which present tuberculous lesions:

The entire carcase and all the organs may be seized—

1. When there is miliary tuberculosis of both lungs.

2. When tuberculous lesions are present in the pleura and peritoneum.

3. When tuberculous lesions are present in the muscular system, or in the lymphatic glands embedded in or between the muscles.

4. When tuberculous lesions exist in any part of an emaciated carcase.

The carcase, if otherwise healthy, shall not be condemned, but every part of it containing tuberculous lesions should be seized—

1. When the lesions are confined to the lungs and the thoracic lymphatic glands.

2. When the lesions are confined to the liver.

3. When the lesions are confined to the pharyngeal lymphatic glands.

4. When the lesions are confined to any combination of the foregoing, but are collectively small in extent.

In view of the greater tendency to generalisation of tuberculosis in the pig, the presence of tuberculous deposit in any degree should involve seizure of the whole carcase and of the organs. In respect of foreign meat, seizure shall ensue in every case where the pleuræ have been "stripped."

The question of the infectivity of milk from tuberculous cows for human beings is a very broad and important one, which has increased since it has been proved that it is not essential that cows should suffer from tuberculosis of the udder for the bacilli to be present in the milk, but that they may also be present when other portions of the body are affected. This in its trail brings in the question of infectivity of milk products. Investigations of Continental authorities, also those of the United States Bureau of Animal Industry, indicate that butter and cheese prove virulent. In one instance 102 samples of butter were examined, and 32·4 per cent. were found to contain tubercle bacilli. Salted butter was found to retain its virulence for six months, and cold storage did not affect the virulence. The timothy grass bacillus was frequently found, as well as the tubercle bacillus.

In cheese the tubercle bacillus survives for a long or short period, according to the mode of manufacture. The experiments indicated that the organism might be dead in as short a period as thirty-three days, or it might be virulent as long as 261 days.

People cannot escape the risk of infection by eating margarine, as in one investigation out of ten samples examined eight were found to be infected.

In conclusion, attention may be called to another aspect of the disease, and that is the fact of cases being on record where it has been found that tuberculous persons and tuberculous dogs, cats, and parrots have occupied the same dwelling.

JOHNE'S DISEASE

Synonym.—Specific Chronic Enteritis of Cattle.

History.—This disease was first described in an article in a German paper by Johne and Frothingham in 1895, and next by Markus in 1904. Since that date it has been found to be widely distributed over the continent of Europe, the British Isles, and North America. It has, presumably, been introduced by cattle into South Africa. To what extent it exists is not at present known; the writer has only met with the disease in one instance.

Nature.—The disease is a contagious chronic inflammation of the intestines, almost exclusively confined to cattle. A case in a sheep was described by Stockman, and a few cases by Sir John MacFadyean in deer in a park. It has not been found possible to transmit the disease to any of the small laboratory animals; guinea-pigs, fowls, rabbits, and pigeons are highly immune.

The disease is caused by a small acid-fast bacillus that greatly resembles the tubercle bacillus, and, like it, is not completely decolourised in forty-eight hours by a 25 per cent. solution of sulphuric acid. One is safe in assuming that this organism is obligatory, but there is not sufficient clinical evidence to show whether the disease arises sporadically. The main seat of multiplication of the organism is the mucous membrane of the intestines. Apparently the small intestine is the primary and most constant seat of invasion, and the part where they are most readily found is the terminal portion of the intestine.

A few of the organisms pass out with the fæces, and it is believed that this is the common method of the spread of the disease naturally—*i.e.*, by ingestion. The disease has been experimentally conveyed by feeding and by intravenous inoculation.

The disease is insidious in character, and herein lies a great danger to the stockowner, as in naturally contracted cases no symptoms may be seen for a year or more.

Symptoms.—The first symptom to be observed in this disease is a loss of condition, usually followed or accompanied by diarrhœa or a looseness of the bowels. Despite a good appetite and a plentiful supply of nourishing food, the animal remains in an un-

thrifty condition with a staring, harsh coat. The loss of condition may be progressive, and if accompanied by a profuse diarrhœa a fatal termination may quickly follow. Frequently, however, affected animals remain in a fluctuating condition for many months, with occasional relaxation of the bowels, and help to swell the ranks of animals known as "wasters" or "piners."

Illness or severe systematic disturbances are liable to bring the disease out in an acute and fatal form in animals not suspected of being affected with it. Cows apparently quite healthy up to the time of calving suddenly develop an acute diarrhœa, waste rapidly away, and die in the course of a few weeks.

The temperature varies; sometimes it is found to be normal, at others it is elevated several degrees. Anæmia is frequently well marked, the mucous membranes and the skin assuming a very pale or livid hue.

Diagnosis.—Men experienced in the disease can make a fairly certain clinical diagnosis, but difficulty exists in distinguishing, without the tuberculin test, between this disease and tuberculosis. In the latter diarrhœa is rarely present; in the former it is usually so. The two diseases are frequently met with at the same time in the same herd, and may exist in the same animal.

A microscopical search for the organism in the fæces usually proves futile, but more success attends such an examination when a finger-nail scraping of the mucous membrane of the bowel by inserting the hand in the rectum is obtained. Failure to demonstrate the bacillus by this means does not justify a negative diagnosis.

Endeavours have been made, so far without any great success, to utilise the tuberculin test—the avian strain of the disease—as a diagnostic agent in Johne's disease, and owing to the possible coexistence of the diseases the method does not appear to hold out very great practical possibilities.

An animal suffering from Johne's disease will seldom, if ever, give a normal reaction to the tuberculin test.

Post-mortem Appearances.—The naked-eye appearance in advanced cases is reliable: the bowel wall is thickened to four or five times its normal thickness, lies in folds, and has a crinkled appearance; it may be pale in colour, and in the majority of cases is so. In other cases it is slightly congested.

The small bowel, particularly that portion where it joins the large bowel, is most frequently affected, but the large bowels may also, and frequently do, show thickening caused by the disease.

In all such cases a bacteriological examination should be made

by gently washing a portion of the affected bowel and making smears from a scraping of the mucous membrane.

Treatment.—It is doubtful if recovery from the disease ever occurs, though it seems probable that some animals possess a considerable degree of resistance, and that mild non-fatal cases do occur; but in animals exhibiting clinical symptoms the disease may be said to be fatal in about 100 per cent. of cases.

In cases taken in hand in the early stages of the disease medicinal treatment, to check the diarrhœa, and highly nutritious food may afford a temporary respite, which may last a year or more, but in the end the result is the same.

In view of the fact that an animal affected with the disease is a source of spreading the malady, the wisdom of keeping it alive is questionable, unless it be for the reason of obtaining a calf, when it should be isolated and all litter, etc., soiled by its discharges should be burnt or ploughed into arable land, or otherwise dealt with to prevent it coming in contact with healthy animals. Disinfection should also be thoroughly employed.

In attempting to eradicate the disease from a herd, one is faced with the difficulty that animals to all appearance healthy may be affected and spreading the disease. Another, so far as we in South Africa are concerned, is the lack of knowledge as to how long the organism can live outside the animal body; whether the disease will be able to exist here to the extent it does in other countries where cattle are stabled to a much greater extent than they are here; and whether the open-air life that the majority of our cattle lead, coupled with a dry climate and hot sun, will not be detrimental to the life of the organism outside the animal, and prevent the contamination of the pasturage for any length of time, and so check the spread of the disease.

A limited experience apparently indicates that amongst animals kept to a great extent in stables the disease in South Africa spreads just as much and is equally fatal to what it is in Europe.

LAMZIEKTE

Synonym.—Gal Lamziekte.

History.—Lamziekte, as it is now called, has been known in South Africa for about fifty years, during which time it has considerably extended its range and become a most serious menace to the cattle industry over large areas in the Union of South Africa, particularly in British Bechuanaland.

Up to the present the disease has not been diagnosed in Rhodesia or other parts of Africa. In Tasmania, a disease of cattle known as " midland cattle disease " is believed to be identical with lamziekte, and it is probable that, in course of time, the disease may be found to exist in other parts of the world.

Towards the end of the nineteenth century the disease, by its ravages, attracted a good deal of attention in Cape Colony, and was investigated by the Veterinary Department of that territory with a certain amount of success. In 1894 Dr. Hutcheon wrote, " The principal predisposing cause is a deficiency of phosphates or bone-forming material in the vegetation where these diseases occur"; and although recent investigations have gone much further and indicate a specific organism as the actual cause of the disease, Hutcheon's findings of the predisposing cause and his preventive treatment of supplying the animals with bone-meal have been confirmed.

In the early part of the present century the prevalence and extension of the disease into localities and farms previously free from it led to great alarm amongst cattle-owners, and to comprehensive experiments and investigation by the Veterinary Division of the Union, to whose reports we are indebted for our present-day knowledge of the disease, a recent report by Sir Arnold Theiler bringing the matter up to date so far as the investigations have gone.

Nature.—Lamziekte, strictly speaking, is not an infectious or contagious disease, yet the unburied carcase of an animal dying of the disease might or might not set up a fresh centre of infection in a locality hitherto free from the disease. Whether it happened or not would apparently depend upon whether the organism

causing the disease was present in the locality as a normal soil saprophyte or whether the climatic and other conditions were suitable to its existence when introduced by the animal carcase, both of which are subservient to the existence in cattle of that form of pica (depraved appetite) known as osteophagia, or the craving for bones. Experiments indicate that for animals to acquire the disease naturally they must possess this depraved appetite and eat decomposing bones or other animal matter containing the organism.

The investigations do not preclude the possibility of vegetable matter also furnishing a suitable medium for the toxic existence of the organism, but as it is an anaerobe (only grows in the absence of air), suitable conditions for its existence in vegetable foods consumed by animals would be rarely, if ever, present under ordinary circumstances under which cattle are kept or fed.

The nature of the disease is that of a ptomaine poisoning: the toxin which poisons the animal originates principally, if not entirely, in the carcases of animals that are left to decompose on the veld. The putrefying carcase of any animal in a lamziekte area, apparently, may be a source of infection when a portion of it is consumed by another animal. The amount necessary to prove fatal has been found to be small, particularly so with laboratory prepared material when injected subcutaneously or when given by the mouth, but even with material collected from the veld 1 ounce or more has been found sufficient to induce the disease in cattle. It follows that only animals with very depraved appetites, in which the craving is great, would eat the more objectionable and highly infected portions of a carcase; but others, by consuming the less objectionable bones in larger quantity, absorb sufficient poison to produce the disease.

The disease has been experimentally produced in a variety of animals, including horses, sheep, goats, rabbits, guinea-pigs, ostriches, and ducks. In nature the disease is rarely seen in animals other than cattle; odd cases do occur in donkeys, goats, poultry, and ostriches. The reason is explained by the fact that animals, other than cattle, do not develop this bone-craving appetite; poultry and ostriches may pick up fragments of toxic substances, and goats and donkeys, it is said, have been observed eating the stomach contents of carcases, which probably accounts for the cases that occur. Sheep are very susceptible (experimentally) to the toxin, and rats appear to be very resistant.

The disease is to a great extent seasonal in character. The

hot, dry weather of the early summer, when putrefaction is rapid, favours the growth of the organism in the carcase; the cold, dry weather of winter tends to prevent to a certain extent the development of toxin in the carcase. Heavy rains, apparently, render the carcase less toxic, and, by a strange coincidence, the seasonal and climatic conditions that govern the toxicity of the carcase also govern the osteophagia of the cattle. In the hot, dry weather, when the toxicity of the carcase is greatest, bone-craving is also at its height.

Spread of Infection.—In spreading the infection of the disease, investigation and experiments have shown that four common modes may be regarded as important: blowflies for short distances, and dust, birds, and moving animals for longer distances.

Blowflies themselves apparently act as a mechanical means of conveying infection from one carcase to another. Experiments have proved that the larvæ and pupæ of the blowfly are toxic, and the former undoubtedly help to distribute the bacteria through the carcase.

The experimental placing of non-infected carcases on the veld in the winter when blowflies are not present has demonstrated that they are not essential to distribution, and it has been assumed that infection from soil and dust occurs. Birds, dogs, and jackals, by carrying portions of the carcase, may distribute the disease to considerable distances, and spores of the organism may also be distributed by means of their fæces.

Research indicates that the bacteria only produce disease when they have grown upon organic matter, and that the animal is able to get rid of other non-toxic bacteria with which it may have become infected; but if such an animal dies from some other cause than lamziekte, outside a lamziekte area, the carcase may become toxic through the invasion of it by the bacteria in its intestines. It is not known how long such bacteria may remain in an animal after it has been removed out of a lamziekte area, nor to what extent it may scatter spores by its fæces.

Pica, or Depraved Appetite.—Depraved appetite in cattle is not peculiar to South Africa. In other countries, the same as occurs all over South Africa, a small percentage of animals are found that will eat various kinds of rubbish; this craving is known as "allotriophagia," but over large areas of South Africa a considerable percentage of cattle exhibit a craving for bones (osteophagia). This craving is one of degree. In one place it may be very mild, the animals only picking up bones if by chance they come across them; in other places the craving is greater, the animals searching

for bones and becoming a nuisance around the homestead by the persistency of their search. The extent to which this craving may exist in a herd may be from 1 or 2 per cent. to practically every animal. The degree to which it is present in the individual also varies; a mild craver will only chew bleached sweet bones, whilst some of the worst cravers will consume bones in a state of putrefaction most objectionable to the palate and sense of smell.

The cause of this craving is apparently a deficiency of phosphates in the food. A series of analyses has shown that the craving is greater when the natural grass upon which the animals live contain the smallest amount of phosphates, and that after a rain in the early summer, when the new young grass contains a greater percentage, the amount of craving in a herd decreases. Craving can also be stopped by feeding the animals on food rich in phosphates, such as wheat, bran, or by the administration of drugs containing phosphorus that can be assimilated by the animal, or supplying the animals with a ration of sterilised bone-meal. Although the investigations, so far as they have gone, point to the foregoing as the cause, the investigators do not wish to bind themselves to such a simple explanation, as it is possible that other factors may produce a similar result, one of which —that is regarded with suspicion—is a high ratio of lime to phosphorus in the mineral matter of the food.

Because " osteophagia " is present to a considerable extent in a locality, it does not follow that lamziekte must necessarily be there—in fact, in some areas it is not present. Investigations, on the other hand, indicate that the organism causing lamziekte may be present in a locality, but owing to the non-existence of bone-craving amongst the cattle, the disease does not occur. In a lamziekte area the appearance of pronounced bone-craving may almost be taken as a premonitory symptom of the disease, but wherever it occurs to any great extent, in view of the recent knowledge obtained it should be regarded with a certain amount of apprehension.

The Causal Organism.—The causal organism has not yet been isolated in pure culture. In impure toxic cultures, which are easily obtained, three different sporulating organisms are constantly present. Two of these, when taken alone, produce no toxin.

Incubation.—In experimentally produced cases the average incubation is about five days, but an animal can be killed almost at will by varying the dose in anything from seventeen hours to ten days. In naturally contracted cases the same rule of quantity

and virulence of the material consumed would follow, but when animals are taken from a non-lamziekte to a lamziekte farm some months usually intervene before such animals exhibit symptoms of the disease. This can be partly explained by the time required for the animals to develop osteophagia to an extent causing them to eat carrion containing the toxin and to the somewhat seasonal nature of the disease.

Symptoms.—As a rule the onset of the disease is sudden. In animals that are kept under close observation premonitory symptoms of an indefinite character may be observed for a day or two. The animal may exhibit a certain amount of uneasiness, and a keen observer, accustomed to the animal, will be convinced in his own mind that something is amiss with it, although unable to explain his reason for thinking so. For purposes of comparison the disease may be divided into peracute, acute, and subacute lamziekte; but there is no definite dividing-line between these forms, excepting that the symptoms develop more rapidly and that death takes place sooner in one form than the other.

Peracute.—In this form of the disease death takes place very rapidly, the common experience being to simply find the animals dead on the veld, never having noticed them being sick. This sudden form of death, so much resembling deaths from anthrax, has caused a lot of confusion in the lay mind, and resulted in wrong conclusions being arrived at by stockowners in many instances. When the animals are noticed to be sick, death takes place in a few hours. The symptoms are very marked muscular weakness, the animal being unable to rise almost immediately after it is noticed sick. Only a small percentage of the cases of lamziekte that occur are of this nature.

Acute.—This is the common form of the disease. The animal will be noticed to be dull and off its feed. In the early stages muscular weakness will be only slight; when it is driven, stiffness will be noticed in the fore-quarters, the animal moves with a sluggish gait, and shows a tendency to lie down. As the muscular weakness becomes greater the tendency to lie down becomes more pronounced. The temperature in all cases of lamziekte remains about normal throughout the disease. The muscular weakness is progressive; the hind-quarters become affected as well as the front; movement is very difficult, and soon, in the course of a few hours or days, the animal is no longer able to maintain the standing position, goes down, and is unable to rise or stand if it is lifted up.

The muzzle becomes dry and caked with dry crusts, and mucus-

covered fæces is passed in small quantities. In some cases paralysis of the throat and tongue occurs. In such the tongue appears limp, the jaws are constantly on the move, and profuse salivation takes place.

For a time, after it goes down, the animal is able to assume the natural position, resting on its breast, but becomes too weak to do so, and falls over on its side; when this occurs, death soon follows. The average duration of this form of the disease is two or three days, and the vast majority of cases prove fatal.

Subacute.—Only a small percentage of the cases assume this form. In it the onset of the disease is not so sudden, and the course is much more prolonged. In the early stages the muscular stiffness in the fore-quarters is only slight, and the muscular weakness increases very slowly. The animal does not appear very ill, it drinks normally and may eat fairly well, or the appetite may be lost. The animal lies down more frequently than one in normal health, but has no difficulty in rising until sometimes from the fourth to the fourteenth day, when it is unable to do so, in nearly all of which cases death follows usually in from two to seven days.

Cases that maintain the power to rise frequently recover.

Post-mortem Appearances.—The post-mortem appearances seen in cases of lamziekte are not very distinctive. Usually the abdominal organs appear to be filled with blood; frequently the lungs are in a similar condition; blood-spots are often seen on the heart, both inside and outside; the mucous membrane of the fourth stomach and intestines is congested, and in subacute cases of the disease this is marked by a muco-enteritis, particularly of the large intestine, the mucous membrane being swollen and wrinkled. The liver is hyperæmic and the gall-bladder frequently distended; the third stomach may contain, in a certain number of cases, dry and compacted food. In the first and second stomach bones, nails, and other foreign bodies are frequently found.

Treatment.—The curative treatment of the disease by medicines may be ruled out as ineffective in practice and wrong in principle, as this disease is one particularly calling for preventive measures. At the same time, some hope is held out that an antitoxin may be found that may be useful for curative purposes; still, this does not alter the fact of prevention being the correct thing to aim for.

In considering the disease, Sir Arnold Theiler gives the following as the etiological chain of six links, and when considering the

prevention of the disease from a practical point of view the case is as he puts it: " The most important link is the one we can break most easily and most cheaply."

1. The toxin which poisons the animal.

2. The toxicogenic saprophytes which elaborate the toxin.

3. The carcase material out of which the toxin is manufactured.

4. The pica (allotriophagia, osteophagia), or depraved appetite, which impels the animal to eat decomposing matter which it would otherwise shun.

5. The vegetation which produces the pica, the soil upon which that vegetation grows, and the climate of the district.

6. The susceptibility of the animal concerned towards pica and towards the toxin.

To the student it will probably appear that the easiest link to break is No. 3, on the well-known principle of " Remove the cause and the effect follows "; clear off all decomposing carcases from the land, and no further cases of disease will occur. Theoretically this is correct, and as a practical measure it is the best to adopt so far as present knowledge goes, but it does not preclude the possibility of other methods being found and that will be adopted either as principal or accessory to it. Where it has been adopted it has given good results. At Armoedsvlakte, the farm in Bechuanaland—which was highly infected, and had a mortality from lamziekte of 30 per cent. in 1914—where the Veterinary Division carried out the majority of their experiments in connection with the disease, the mortality with a herd of 500 cattle was reduced to below 2 per cent. after the present known methods of preventing the disease were adopted in 1919.

As a practical proposition, owing to the farming in lamziekte areas being of an extensive character, the clearing of all carcases from the land is a matter almost of impossibility from an economic point, owing to the difficulty of finding all carcases over such wide areas. The carcases of large animals are, of course, the greatest source of danger; fortunately, they are the easiest to find, and, so far as mature animals of domesticated stock are concerned, they are usually soon missed from the herd, but sheep, lambs, kids, ostrich chicks, etc., dying with the carcases of premature and stillborn calves, foals, etc., have also to be sought. Then there are the carcases of wild animals to be thought of, such as wounded game. Provided all carcases of the larger animals are cleared away, the number of cases of the disease will be reduced to small proportions, but there will still remain carcases of hares, mierkats,

birds, barbels, and other small animals that will not be found and that may cause a small number of cases of the disease.

The next link in the chain that the present preventive measures aim at breaking is No. 4, osteophagia, and the material that is used for this purpose is bone-meal. It is possible that in the near future a cheaper substance may be found to accomplish this object, but at present bone-meal is used, as it is cheaper than feeding animals on a liberal mixed ration rich in phosphorus or suitable drugs containing the same ingredient, both of which reduce the craving fairly quickly.

Broadly speaking, all cattle are fond of bone-meal and will readily consume it, and although it will improve cattle in condition to an extent that the extra price obtained for cattle marketed will more than pay for the cost of the bone-meal consumed by them, and that its consumption by cattle will, to a limited extent, improve the soil, yet it might not be economic in a lamziekte area to give cattle all the bone-meal they would eat.

In connection therewith it may be well to point out that in such areas the cattle are not different from those elsewhere; they breed and arrive at maturity as fat, well-conditioned beasts, and usually it is the animals in the best condition that are the greatest cravers. In consequence, it followed that there was necessity for finding a method of gauging the craving of the animals; this was done by testing cattle with bones in different stages of decomposition, and classifying the animals according to the degree of " rottenness " in which they would consume bones.

For the purpose of this test, the bones that were collected on the veld were used; hence occurred what has been termed the " bone paradox "—bones slay and bones save ! The toxin which produces the disease was found to be thermo-labile—*i.e.*, is easily destroyed by heat. It is destroyed in a few minutes at 80° C., and at 75° C. by more prolonged heating. Therefore, the bones which produced the disease can, after heating, be used to cure it. In selecting veld bones for this test bleached or sweet bones should not be taken, but those having a slightly putrid odour should be, so that only cravers will touch them. They must then be sterilised by boiling or steaming them for one hour on each of three consecutive days. These sterilised bones should still smell putrid, but if properly sterilised will be perfectly harmless. Such bones are then placed in a trough, and the herd is tested by bringing the cattle to them in small lots of ten or twelve, and watching to find out which are the cravers—that is, the animals which will pick up and chew such bones. These are

separated from the rest of the herd for treatment. After the first test the balance of the herd which did not exhibit any craving may be retested periodically—say every fortnight—to see whether any have developed it in the interval; if so, they are removed and put under treatment.

After the bone-eaters have been weeded out of the herd they are put on a daily ration of from ½ to 1 pound bone-meal to each animal; this is best given mixed with a small quantity of bran, to which a little salt may be added, although it is not really necessary. To ensure that each animal gets its full ration of bone-meal, it is advisable to feed them separately, a trough to each beast, which may mean that the animals have to be fed in batches according to the number of troughs available. This ration stops the craving for bones in from one to three weeks; therefore it is advisable to retest these animals, as in the first instance, every week or fortnight. Those that are still cravers continue with the ration; those that have lost the craving are put on a smaller ration of 3 or 4 ounces of bone-meal daily. It is advisable to periodically retest the latter to find out if any have gone back to craving.

If this system is carried out methodically it will be found much less troublesome in practice than it appears at first sight. It is not necessary to feed bone-meal all the year round; it should be given during the hot, dry months of spring and early summer, when craving is at its worst. As soon as good rains have set in it may be discontinued. During the winter, although craving may be present to a certain extent, owing to carcases not being so poisonous, it is not so necessary to feed bone-meal. If an average diligent search for and clearing away of carcases has been made, there will be little chance of animals contracting the disease during winter.

Of course, if it were possible to keep the land clear of all carcases all the time there would be little chance of the disease occurring at any time; therefore it follows that the less carrion there is on the land, the fewer cases of lamziekte there will be.

DISEASES CAUSED BY THE FOWL CHOLERA TYPE OF BACTERIA

In France the term "pasteurella" has been used for the fowl cholera type of bacteria, and "pasteurellosis" to cover the resulting diseases; and though this nomenclature is perhaps not inappropriate, as it was against fowl cholera that Pasteur first used his method of preventive inoculation, these names have not caught on with English investigators.

Hueppe a number of years ago, as a common designation for these clinically different diseases, suggested the term "septicæmia hæmorrhagica," but the name is perhaps not well selected, for several of the maladies included in the group have not hæmorrhagic lesions. This is conspicuously true with fowl cholera, and also with a good many of the others; therefore, although the terms "pasteurellosis" and the "hæmorrhagic septicæmias" are to a limited extent used to designate "diseases caused by the fowl cholera type of bacteria," the latter term, although more cumbersome, more correctly conveys the facts and is apparently coming into more frequent use in the English language.

The bacilli found in the several diseases of this type are morphologically and culturally indistinguishable, but they are biologically different. It is uncertain if these various organisms should be regarded as distinct species or as varieties of the same species. Some are mainly or entirely saprophytic; some may be encountered at any time in putrid or partially putrid materials; some are of common occurrence in the soil and water; and others are the cause of serious diseases.

With bipolar staining organisms (organisms staining at their extremities) indistinguishable one from the other but causing distinct clinical affections, one would expect, if they represented varieties of the same species, that the variety in one species should confer some protection in other types of the disease—that is, that the fowl cholera organism ought to protect against swine plague; but as a rule experiments have failed to show protection of one organism against another, though it has been asserted in some cases that protection has been conferred.

All the domestic animals and several of the wild ones may become infected with diseases due to these organisms. Some of these diseases have been known for a long time. Bubonic plague, due to the *Bacillus pestis*, a bipolar staining bacillus, is the Black Death of the Middle Ages. In addition to the diseases hereafter set out, a great number caused by this type of organism have been described in different species of birds (cage birds in particular) and mammals—in the turkey, goose, duck, rabbit, swan, pheasant, partridge, grouse, pigeon, mouse, etc.

The usual natural method of infection seems to be by ingestion, but it may occur through wounds and abrasions of the skin, and possibly also by inhalation. In some forms of the disease it seems possible that infection may be transmitted by skin parasites, such as lice in typhus, while in bubonic plague the rat flea (*Pulex cheops*) is known to convey the *B. pestis* from rat to rat, and from rat to man.

Fowl Cholera.

Synonyms.—Avian Cholera, Avian Septicæmia, Avian Pasteurellosis, Hæmorrhagic Septicæmia of the Fowl.

History.—The disease is probably world-wide in extent. It has been observed and studied all over Europe and North America. It is of frequent occurrence in South Africa, and has been observed in other countries. It has been recorded from probably as early as 1600 to the present day, during which time a very great number of serious outbreaks of the disease have occurred. In 1877-1878 Perroncito in Italy and Semmer in Russia discovered the causal organism; previous to this its contagious nature and the possibility of transmitting the disease experimentally by ingestion and inoculation had been demonstrated.

In 1880 Pasteur studied the disease, and it was in connection with it that the first bacterial vaccine was invented and elaborated by the great Pasteur, who thus laid the foundation upon which further developments of vaccine therapy have been built. In 1900 the disease was very prevalent in Great Britain, where it was studied by MacFadyean.

Nature.—The cause of the disease is a minute bacterium, an ovoid or bipolar organism, or figure-of-eight bacillus, known as the fowl cholera bacterium or bacillus, also as the *Bacillus avisepticus*, *Bacterium avicidium*, or the *B. choleræ gallinaceæ*. In temperate countries the organism appears to be an obligatory parasite, and there is no evidence of the disease ever being

sporadic. In most cases, when an outbreak of the disease occurs, there is found to be a history of a recent introduction of a fowl or fowls from outside.

It appears probable that in warmer climates the organism may propagate in a saprophytic manner, and the disease may therefore be sporadic.

The disease is a typical septicæmia. In most cases at the time of death the organisms are in immense quantities in the blood everywhere, but the richness varies, though they are never so sparing that they could be overlooked when a microscopical examination of the blood is made. Quite frequently they outnumber the red corpuscles. When distinct lesions are present, the bacteria are very numerous in them; they are also constantly present in the intestines and fæces, but cannot be demonstrated in the excrement. In the blood of the fowl more than 90 per cent. of the organisms are ovoid in shape; they appear to differ in size, especially with the method of staining, and under one method the great majority of the ovoid bodies exhibit bipolar staining. The organism is non-motile.

As a natural disease fowl cholera is most commonly observed in the common domestic fowl. Inbred and highly developed breeds of poultry confined in runs are perhaps more susceptible than the common barnyard fowl, but the conditions under which the birds are kept may possibly account for the apparent greater susceptibility. All common farmyard poultry are susceptible to the disease, and may be naturally attacked. It is readily transmissible by inoculation to a great variety of wild birds; the pigeon is particularly susceptible to the disease.

The common natural method of infection is by food or water contaminated with infected excrement; therefore it follows that the disease may be transported from one place to another by a person's boots; by wild birds; by coops, boxes, baskets, etc.; by the purchase of infected grain and other fowl foods; as well as by the introduction of infected poultry.

The disease can be produced experimentally by ingestion, but inoculation is the more deadly method, and the period of incubation by this method is of shorter duration. Minute quantities injected subcutaneously suffice to induce the disease. After experimental inoculation pigeons generally die during the first or the second forty-eight hours; the period is the same or a little longer in the common fowl; the rabbit is in the highest degree susceptible. Death ensues within twenty-four hours; it frequently occurs within twelve to eighteen hours; therefore, if its

effects are to be seen, it is advisable to inoculate late in the afternoon. To ensure infection it is only necessary to merely prick or scratch the ear with the point of a needle containing a trace of the organism or infected material. As in the fowl, the disease is septicæmia in the rabbit. The richness of the blood in organisms is also a little variable, but usually the number of bacilli present is colossal.

The disease can be transmitted to the mouse by inoculation, but the guinea-pig is very much less susceptible than either the rabbit, fowl, or pigeon; but by passage the bacilli may be made intensely virulent for the guinea-pig.

The disease never extends to the domesticated mammals, but it has been alleged that an outbreak in fowls has served for an outbreak of swine plague amongst pigs.

The death-rate varies from 50 to 95 per cent. of the fowls attacked, according to the virulence of the outbreak.

Symptoms.—The symptoms of the disease are frequently classed as peracute or sudden, acute, subacute, and chronic according to the length of time the affected birds live. There is a similarity in the symptoms presented in all the forms.

Peracute Form.—In this form of the disease the bird may be found dead, or death may occur after the first symptoms are manifested in from a few minutes to a few hours. In the cases seen the bird is sleepy, does not move, the wings and tail droop, the head is brought round under the shoulder, the feathers are opened, and the bird has the appearance of a ball as it stands in one place.

Acute Form.—In this form the foregoing symptoms are not so sudden in their appearance. The bird loses its appetite and activity, great thirst is usually present, the bird frequently stands "like a ball" near the drinking water, and if made to move great weakness is exhibited, the bird staggers and perhaps falls down on its breast. The comb and wattles are purple or black in colour. A profuse diarrhœa is present which soaks and mats the feathers around the vent. The extremities become cold, and the bird dies usually within three days.

Subacute Form.—In this form of the disease it may be noticed that the fæces are passed in large quantities mixed with mucus before diarrhœa sets in; the birds rapidly lose weight and become so weak that the slightest touch causes them to topple over. The symptoms described in the foregoing forms appear, but more gradually, and the bird survives from three to ten days.

Chronic Form.—This form is frequently met with in birds

that have survived the foregoing forms of the disease; it is present to the greatest extent at the end of an outbreak, when the disease has either lost some of its virulence or more probably the surviving fowls possess greater natural powers of resistance to the disease than those that succumbed early in the outbreak.

The symptoms are similar, but less intensified, and consist of dulness, weakness, emaciation, loss of appetite, diarrhœa, standing like a ball, and the mucous membranes pallid.

This form of the disease may persist on infected premises for months.

Post-mortem Appearances.—After subcutaneous inoculation in the fowl, rabbit, or pigeon there is a local lesion at the site of injection; but when death has occurred within twenty-four hours this is quite insignificant, and shows only a little watery œdema of the connective tissue and congestion. If the inoculation was intramuscular, a circumscribed necrotic area is found. In cases of death from inoculation as a rule there are no lesions other than the local ones.

In natural cases there is constantly an enteritis (inflammation of bowels) present. This is the primary condition, but if the intestines are not examined when fresh, there may be no lesions elsewhere to be seen. In a good many cases of the disease there are very minute multiple areas of necrosis in the liver substance, the appearance simulating a fine miliary tuberculosis. In a small number of cases the lung tissue is hepatised. Other post-mortem appearances may be present in different cases, but also may be present in other diseases, and cannot be regarded as diagnostic.

Treatment.—In the acute forms of the disease not much can be done in the way of medicinal treatment, but in those cases that permit of it the following mixture has been recommended: Equal parts of the sulpho-carbolates of calcium, sodium, and zinc. Of this mixture, give ½ grain three times a day to each sick bird.

Perhaps the best preventive treatment, where it can be adopted, is to remove the apparently healthy birds to a fresh site, away from all contamination. Particular attention should be paid to avoid infection being conveyed to the new site by utensils, food, etc., and to prevent its infection by the isolation of any further birds on the first appearance of sickness.

The old site should be thoroughly treated by fire, disinfectants, lime-washing, digging up, planting of crops on site, etc., resting it from poultry for twelve months.

Affected birds will be treated according to the circumstances.

In many cases, to prevent gross infection of the site, it is better to destroy them. All dead birds should be burnt.

Pasteur found that a culture of the fowl cholera bacillus left exposed to the air gradually loses its vitality and dies in about two months, and that the bacteria are attenuated, roughly in proportion to the age of the culture. Taking advantage of this discovery, Pasteur started two vaccines of different strengths, to be used in a similar manner as the anthrax vaccines are used.

The attenuation of the culture exposed to the air does not occur with any regularity, and the cultures readily die out if they are not renewed. They may die in less than a fortnight or a month. The organism does not form spores.

A protective serum can be obtained by hyperimmunising horses with large quantities of the culture.

The Pasteurian method of inoculation has never been tried on a large scale. One of its great disadvantages is that it takes a fortnight to confer immunity; therefore, when an outbreak occurs the majority of the fowls might be dead in less time than that. Perhaps the greatest drawback to preventive inoculation in poultry is its cost compared to the usually small value of fowls.

Rabbit Septicæmia.

Koch applied this term to a fatal condition set up in rabbits inoculated with blood from various sources, especially with blood not taken with aseptic precautions. Such rabbits sometimes died after a very brief interval, and the microscopic examination of the blood revealed the same condition as if the animal had died from fowl cholera, and the blood had the same morphological cultural and staining character.

Since the foregoing observations were made, spontaneous outbreaks caused by a bacterium of the fowl cholera type have been observed amongst both wild and domesticated rabbits. The only distinction between the organisms is that neither the tame nor wild rabbit one is pathogenic, or very much less so for the fowl or pigeon than the fowl cholera bacillus is.

The occurrence of such spontaneous outbreaks is very difficult to account for. It is difficult to believe the organism is constantly present amongst rabbits.

Septicæmia Hæmorrhagica in the Ox.

This disease was apparently first recognised by Bollinger in 1878 amongst the animals in the Royal Park at Munich. It affected the deer and the wild swine, and spread to the cattle, pigs, and it was said also occasionally to horses. The disease was investigated by Kitt, who discovered the causal organism.

Since then what has been held to be the same disease has been observed in South-Eastern Europe, Northern Africa, and Asia.

Clinically the disease bears a close resemblance to anthrax, and it is thought that it was generally roughly diagnosed as anthrax previous to the discovery of the organism. The period of incubation is short, and the duration of the illness is no longer than in anthrax, but sometimes the animals survive as long as a week. In cattle in a very large proportion of cases there is during life a marked swelling of the tongue and throat, and it appears possible that the disease described in the old textbooks as "gloss anthrax" was this disease; it is quite certain that tongue lesions are seldom, if ever, met with in anthrax or quarter evil. The post-mortem appearance of the lesions is not distinguishable from anthrax in the pig. Inflammation of the stomach and intestines is present, but the spleen is said not to be swollen; there may be exudations into the chest or abdominal cavities, and blood extravasations may be present.

The disease exhibits a seasonal occurrence, being most frequently encountered in the summer-time. It is fatal in 80 to 90 per cent. of cases. It is transmitted by inoculation and ingestion, and it is supposed that the bacterium exists mainly as a saprophyte.

Barbone.

Barbone is the name given to a similar disease that affects buffaloes. It was first described in Italy, and since has been found to be of common occurrence in South-Eastern Europe and Egypt, and has also been seen in India.

The disease bears the closest resemblance to the bovine disease. There are conspicuous throat lesions, and the disease is just as fatal. The habit of the organism is believed to be saprophytic.

In both diseases diagnosis can be made in fresh carcases by microscopical examination of the blood, which contains large numbers of bipolar staining bacteria of the fowl cholera type. These organisms are also to be found in the lesions.

Cornstalk Disease.

This disease is one affecting cattle in North America, and derives its name from the fact that it was believed to be contracted during the time the animals were turned into the lands to eat the maize stalks (mealie stalks in South Africa, cornstalks in America).

The disease was investigated by Billings, and proved to be due to an organism of the fowl cholera type.

The symptoms in the affected animal are somewhat similar to those seen in an animal suffering from contagious pleuro-pneumonia or lung-sickness, and the post-mortem appearance of the lungs may be said to be practically indistinguishable by naked-eye examination. It was this latter character that brought the disease into great prominence between 1879 and 1891, when a considerable number of living cattle were imported into Great Britain and France from North America, of which a number were found to be suffering from a disease of the lungs that was diagnosed by the officials of the English Board of Agriculture as contagious pleuro-pneumonia, but by Williams in Scotland and Nocard in Paris as broncho-pneumonia. A great controversy arose which led to a fuller investigation of the disease both in America and Europe. For full information refer to Williams's "Veterinary Medicine."

Septic Pleuro-Pneumonia of Calves.

This disease was first described by Poels in Holland, and it has occurred in various parts of the continent of Europe and the British Isles, and precisely similar cases are met with all over the world, including South Africa.

The disease may occur in isolated or sporadic cases, but as a rule it presents itself as a contagious pneumonia in quite young calves, and is not often seen in calves after they are about five months old. It is possible that the habit of the organism may be saprophytic, but once the disease has started in a troop of calves it exhibits a markedly contagious character.

The disease is very fatal and frequently very rapid in its course in the first animals attacked. Many calves succumb during the first two or three days of illness; others linger on for a longer time; some ultimately recover, but are usually stunted and unthrifty. The symptoms presented are loss of appetite, rapid wasting away,

short husky cough, and evidence of acute pneumonia. The post-mortem appearances are extensive croupous or hæmorrhagic lesions in the lung tissue and "patchy" inflammation of lungs. In cases that live for some days these patches may have become gangrenous and contain pus. Pleurisy is also present, and may be accompanied with an effusion into the chest cavity. In very young calves cases are met with that are septicæmic in their nature, and in which the blood swarms with organisms of the fowl cholera type. It is thought probable that the infection in such cases may have occurred via the navel.

The method of infection is not known, but it is probably by ingestion. The disease persists on infected premises.

Not a great amount of success can be claimed for any curative measures that have been tried. Cures have been claimed for the subcutaneous injection of tallianine, and relief has been given by inhalations of steam medicated with carbolic acid.

The treatment indicated and that has been found of the greatest success is of a preventive nature. The calves should be kept in a good thriving condition and free from parasites, particularly external ones such as ticks and lice. Causes which tend to lower the vitality of the animal may be regarded as predisposing to the disease. Sick calves should be immediately isolated from the healthy ones, and particular attention should be paid to thorough disinfection of the calf-pen, the bedding, and everything that may have become contaminated by contact with the infected animal.

In Sheep.—A similar disease exists in sheep, which attacks adult as well as young animals. Further research work is required in both these diseases.

Swine Plague.

Synonyms.—Pig Pneumonia, Swine Septicæmia (French, *Pasteurellose du porc;* German, *Schweineseuche;* American, Septicæmia Hæmorrhagica).

The term swine plague was introduced into the English language from the German *Schweineseuche,* and the principal literature on the disease is in German. In Germany and America this disease has for years been represented as a most serious contagious disease of pigs, and is responsible for immense losses. It occurs sometimes as an acute septicæmia, with the bipolar organisms present in the blood, and inflammation of the stomach and bowels revealed on post-mortem examination; but the common form of the disease is an acute or subacute pneumonia,

with extensive hepatisation of the lung tissue, in which bipolar organisms are found in great abundance.

In Germany until a few years ago the disease was regarded as highly contagious, usually pneumonic in form. Chronic cases were also seen in which the pneumonic lesions underwent a sort of caseation. Later on the German authorities modified their views somewhat as to its nature, but held that it was practically always contagious.

It has always been admitted in Germany that the so-called outbreaks of swine plague are in a large proportion of cases complicated with swine fever, and that pulmonary and intestinal lesions occurred simultaneously; this was also observed to be the case in the United States. It was never possible to reconcile the experience with regard to swine pneumonia in Great Britain with the German account of swine plague. There it is not uncommon to have fatal pneumonia in the pig corresponding with swine plague, and the lesions swarming with ovoid bacteria agreeing with the bacterium held to be the cause of German swine plague; but the remarkable discrepancy is that while this swine pneumonia is common in Great Britain, there is no evidence that it propagates by contagion, and it is doubtful if cases ever occur independent of swine fever.

Bacteria indistinguishable from the bacteria of swine plague can be found in the mouth and air passages of healthy pigs, and the most reasonable interpretation of the facts of swine pneumonia in Great Britain is that as a rule it is a simple complication of swine fever caused by a bacterium which is frequently or always present in the mouth or air passages of pigs, and which is ordinarily devoid of pathogenic power for the pig; but that when a pig becomes the subject of swine fever its defences are weakened, and in consequence of this the bacterium begins to propagate in the lung tissue and sets up a pneumonia. No other interpretation of the facts enables one to understand why the two diseases are so constantly associated.

In Germany attempts have been made to control the disease by inoculation, and have been carried out on a very large scale; but it was admitted that these protective serums did not give very good results. This was attributed to the possibility that the swine plague bacterium might vary from outbreak to outbreak—that is, that there were various strains of the bacterium, and that a serum obtained by hyperimmunising a horse from a particular strain would only be good for that particular strain. A polyvalent serum was then prepared by using for the hyper-

immunisation of the horse a large number of different strains, and at first it was declared that the method was successful; but at the present time the idea that swine plague, even in Germany, is a highly contagious disease has been abandoned, and it is admitted that it occurs there, as in Great Britain, usually as a complication of swine fever.

The bacterium found in swine plague is quite indistinguishable morphologically and culturally from the fowl cholera bacterium, but its pathogenic power varies for different animals. By ingestion it is not pathogenic to the fowl, and is not markedly so by inoculation. Its virulence for the rabbit is very variable, and as a rule after inoculation some days elapse before death occurs. It is not highly pathogenic to the pig experimentally by ingestion or inoculation; by ingestion the results are often negative.

SPECIFIC OPHTHALMIA IN THE HORSE

Synonyms.—Contagious Ophthalmia, Infectious Ophthalmia, Periodic Ophthalmia.

Definition.—By specific (that is, definite and distinct) ophthalmia in the horse is meant a disease, believed to be infectious, the cause of which is unknown, but is probably a bacterium, and results in blindness after one or more attacks.

History.—The disease first came into prominence during the Boer War, 1899 to 1902, during which many horses became affected. Little was heard of the disease after that event, although odd cases have been observed, until the recent Great War, when again a small percentage, probably under 5 per cent., but which amounted to a large number in the aggregate, were disabled by this disease.

Very little is given in the English textbooks on the malady, and outside war very little appears to be known of it. It is reported to have been recognised on the continent of Europe for a number of years, and it is possible that under the name of " moon blindness " the disease was present in Great Britain some fifty or more years ago. It has been known in America for at least fifty years, and it was thought to be more frequently met with in horses from that continent than from other places during the Great War.

Nature.—Specific ophthalmia is an inflammation of the eye that is not confined to any particular portion of it; all sections share in the change in some degree. The disease is of a recurring nature, a few weeks usually intervening between the attacks; in exceptional cases several months may intervene. The termination of the disease is in almost all cases blindness, which may result after the first attack, but such a case is exceptional. The rule is for the horse to go blind after three or four attacks, but seven or eight may occur before blindness results.

In 1918 Captain Dalling claimed to have discovered a bacillus in the optic nerve which he regards as the cause of the disease. He further stated he had no difficulty in making cultures of it;

he also prepared a vaccine with which he claimed to have had good results.

Numerous attempts to transmit the disease experimentally from horse to horse were made during the Great War, but no one was able to do so. The material used was taken from diseased eyes, but most of this work was done prior to Dalling's discovery.

In America the disease is popularly believed to be hereditary, and it has been stated that nearly all the progeny of a stallion suffering from specific ophthalmia will become blind between the ages of four and seven years. A popular belief such as the foregoing may be taken to indicate that at least there is a hereditary predisposition to the disease.

The disease usually occurs sporadically, but shows no tendency to spread by direct contagion from one horse to another. The collection of horses together in great numbers would appear to be a predisposing cause, and with regard to its infectiousness it has been remarked that the disease has never been observed in Australia, yet in both the Boer and the Great War many Australian horses were affected.

Symptoms.—The first symptom noticed usually is that the eye is closed with tears running down the cheek. One or both eyes may be affected; perhaps in the majority of cases the disease commences in one eye, and subsequently both eyes are affected. There is a certain amount of conjunctivitis (inflammation of the mucous membrane of the eye) present, with distinct photophobia (fear of light); the pupil of the eye is contracted. A discoloration of the eye is commonly present; it appears as a yellowish-brown tint due to a precipitate in the anterior chamber, which falls to the lower part, and may appear only as a thin line or it may cause a cloudiness extending half-way up the eye. The bloodvessels of the cornea (front of the eye) are congested, particularly noticeable at the periphery. This congestion is quickly followed by a smokiness and opacity which may extend over the entire surface of the cornea. It is the rule for these symptoms which appear on the first occasion to become more marked in subsequent attacks, but in a certain number of acute cases the animal is left blind by a single attack of the disease.

The blindness may result from a number of conditions within the eye, the most common one, perhaps, being that the crystalline lens becomes opaque; adhesions may take place between the iris (the curtain that forms the pupil of the eye) and the lens. In other cases the lens becomes dislocated, or deposits may form in the posterior chamber of the eye. Frequently the cornea

and the front of the eye are very little altered, but owing to some pathological change at the back of the eye the animal is totally blind. A matter of considerable importance is the ability to detect the disease in an animal that has recovered from an attack. During the South African War it was observed that an animal that had suffered from it had frequently a slight but peculiar wrinkling of the skin above the affected eyes. An affected eye appears smaller than the normal eye. On examination of the internal structure of the eye, the iris is found to have lost some of its lustre and is frequently somewhat ragged around the pupil.

Treatment.—No specific treatment of the disease is known. The treatment employed is of a palliative nature. The first thing to do is to keep out the light by shading the eyes; this can be done by attaching something, preferably of a dark colour, to the headstall. Fomentations with hot water seem to give relief, and the drug that has been used to a greater extent than anything else is atropine; this is used in the proportion of 4 grains of atropine to 1 ounce of distilled water, or 10 grains of atropine to 1 ounce of vaseline. Many practitioners prefer the ointment to the liquid. Whichever is used is applied once or twice a day.

Upon the whole, opinion is adverse to the beneficial results following the injection of 1 c.c. of Lugol's solution of iodine, which is nearly a specific for ophthalmia in cattle (for instructions regarding application see Specific Ophthalmia in Cattle). Specific ophthalmia in horses and specific ophthalmia in cattle are distinct and different diseases, which probably accounts for the difference in the results obtained; but it should be pointed out that some practitioners hold that the iodine treatment clears the eye up in horses, although the disease recurs again, the same as it does after any present known treatment.

SPECIFIC OPHTHALMIA IN CATTLE

Synonyms.—Ophthalmia, Eye Disease.

Definition.—Specific ophthalmia in cattle designates a disease of the eye that is apparently contagious, but the cause of which is unknown.

History.—The disease has been recognised in South Africa for a number of years, and, when present, it attacks practically all the young cattle on the farm; it may also attack mature cattle, but not so frequently as younger animals.

In Rhodesia prior to the advent of the cattle-dipping tank this disease was one of the greatest bugbears of the cattle-owner, but with the regular and systematic weekly dipping of cattle the disease decreased considerably, and from some herds it disappeared entirely. The beneficial effects following dipping may have resulted from the dipping tank being situated usually near the homestead and the cattle kraal, killing a great number of the transmitters of the disease in the shape of moths, flies, etc. The disease has by no means disappeared; recrudescences of it occur, some seasons appearing to favour it, when a number of cases are in evidence.

Nature.—The actual cause of this disease has never been scientifically determined. The ordinary pus-forming organisms have been found present in cases, and it is possible that it may be due to one or other of these.

The transmission of the disease from sick to healthy animals appears to be a mechanical one, and insects that are in the habit of sucking the natural moisture from the lids of the eyes of cattle are probably a great source of the spread of the disease in a herd.

Perhaps there are more than one species of insect involved in the transmission of the disease. The one that is most frequently accused of transmission is the white moth that clusters in great numbers around the eyes of cattle in the evening. There is also a brown moth of fair size which has similar habits, but flies and gnats also attack the eyes and may be equally guilty.

The actual loss to such owners in dead animals is not great, but there is a considerable loss in depreciation in value by affected animals being stunted and failing to grow out, by odd animals going blind in both eyes, and by others losing the sight of one eye. Even if the animal is not entirely sightless in the affected organ, the eye has probably a spot on it or other disfigurement; but the greatest consideration to most cattle-owners is the worry of seeing their animals suffer, the time expended on dressing the eyes, and the very slow and inappreciable benefits attending their efforts.

Symptoms.—The symptoms of the disease are an intolerance to the light in one or both eyes, usually one. The eyelids are kept closed, and a watery discharge from the eye is present. On examination, the eye presents an inflamed condition, and in the majority of cases a small ulcer about the size of a kaffir-corn seed is observed in the front of the eyeball, usually directly in the centre. After a time, if no remedies are applied, the inflammation subsides, the watery discharge stops, and the eyeball assumes

a milky opacity with loss of vision. The ulcer may have healed up, and gradually the opacity of the cornea may disperse and the animal to a greater or less extent recover its sight. On the other hand, the ulcer may have increased in size, a dirty discharge oozes from the eye, and finally the eyeball bursts and discharges its fluid contents. The outer coverings of the eye gradually heal after this rupture, but leave the animal entirely blind in that organ. In other cases the pressure set up by the eyeball may not quite cause a bursting of the cornea, but only causes it to bulge out at the point where it has been weakened by ulceration. This bulging may become organised with bloodvessels, and assume the appearance of proud flesh, and require touching with caustic to reduce it. A frequent sequel to the disease is for the eyeball to remain enlarged and of a milky-white appearance for many months, of course with a permanent loss of sight.

Treatment.—The treatment of the disease consists of keeping the eyes clean and applying one or other of a variety of applications. One of the favourites is corrosive sublimate (1 to 1,000 in water) dropped into the eye. Another is a saturated solution of boracic acid—that is, as much boracic acid as water (boiled and then cooled for preference) will dissolve; about 1 ounce to a pint makes a useful eye lotion. Another line of treatment is by blowing a powder such as boracic acid or calomel into the eye. Still another method is to use yellow mercurial ointment, and some good results are claimed for this latter treatment. My experience leads me to the conclusion that better results and quicker are obtained when one particular line of treatment is not followed for too long a time, and that it is advisable to switch off from one on to another every few days, change from powder to ointment or lotion, or vice versa; but the great objection to all these treatments is that they have to be applied daily, and the slow progress made.

The native treatment for this disease is to burn a ring on the skin about 2 or 3 inches from and all around the eyeball. This treatment is apparently successful in many cases, but is drastic and disfiguring.

Another treatment that is said to be effective is to dust ordinary red lead into the eye every fourth day.

A treatment that has been found to be almost specific in the hands of many operators is the injection of 1 c.c. of Lugol's solution of iodine into the pad of fat that lies at the back and at the top of the eyeball. The operation is as follows: Disinfect

the upper eyelid, then insert a thin hypodermic needle through the skin above the centre of the eye between the eyeball and bone (supra-orbital process), taking care not to injure or penetrate the eyeball when doing so. Press the needle in to a depth of $\frac{5}{8}$ to $\frac{3}{4}$ inch. The syringe is then attached to the needle and the solution injected.

After the injection of the iodine local swelling occurs, sometimes closing the eyelids for a couple of days. Great improvement follows the treatment, and usually only one injection is required, but a second one may be given if required six to eight days after the first.

CATARRHAL ROUP

Synonyms.—Roup, Wet Roup, Avian Contagious Catarrh, Fowl Distemper.

Nature.—Catarrhal roup is a contagious disease of birds, especially of the fowl. The term "roup" is of Scandinavian origin, denoting hoarseness, and is generally used by poultry breeders to designate any catarrhal affection of the head. Whether catarrhal roup and diphtheritic roup are distinct or merely different forms of one and the same disease is still a matter of some uncertainty. The causal organism or organisms have not yet been satisfactorily determined—in fact, more than one organism has been found to be present—but the weight of opinion, perhaps, indicates an ovoid bacillus of the fowl cholera type as the cause of the disease.

Symptoms.—These are exhibited in varying intensity in different cases. The affected bird is more or less dull, off its feed, sneezes, and has a hoarse cough. The mucous membranes of the eyes and nostrils are inflamed, and a discharge is present which is at first watery, but later on becomes muco-purulent, and the bird has an offensive smell about the head and mouth. Providing these symptoms do not increase in intensity, recovery usually takes place.

In other cases the onset of the disease is marked by greater constitutional disturbance of the affected bird, the dulness and prostration are more marked, the eyelids are glued together or the swelling of the mucous membrane causes the eyelids to bulge out and cover the eyeball, and the bird cannot see to feed or move about; the sac under the eye becomes filled with secretion and distended. The discharge completely blocks up the nostrils, causing the bird to breathe through the mouth, accompanied by a croaky noise and frequently by the emission of a foul watery discharge from the mouth. Emaciation in this form of the disease is rapid, and the death-rate is high.

Treatment.—The disease is difficult to eradicate once it has started in a poultry run. Cold, damp weather and draughts favour its development; therefore as far as possible these effects

on the birds should be guarded against. The affected birds should be isolated in a warm, dry place and fed on hot foods, the eyes and nostrils sponged two or three times a day with corrosive sublimate (1 to 1,000 in water), and a pill composed of the following ingredients should be given twice daily:

Take of strychnine 1 grain; sulphate of quinine 15 grains; powdered capsicum 30 grains; extract of gentian sufficient to make into thirty pills.

Disinfection of the run and everything that may have come in contact with the discharges from the infected birds should be carried out.

DIPHTHERITIC ROUP

Synonyms.—Avian Diphtheria, Avian Roup.

Nature.—It is a contagious disease of birds, characterised by a diphtheritic inflammation of the mucous membranes, particularly those of the head. The disease has been seen in a great number of birds, both wild and domesticated, as well as in all the different species of farmyard poultry, but the domestic fowl and the pigeon are the most common subjects of the disease. It has been observed as an epizootic amongst wild pigeons.

The disease is world-wide in occurrence, and is commonly present in South Africa. Various organisms have been isolated, including one of the fowl cholera type and another very small organism by Bardet, but at the present it is uncertain which organism is the actual cause of the disease. It is a highly contagious disease, and an outbreak in a poultry run always causes a heavy loss. The incubative period is probably about a week.

Symptoms.—Usually the disease is well developed in the first birds affected, and has got a good hold of the flock before it is discovered. The first symptoms noticeable are the sick bird stands about with wings and feathers drooping, the head becomes purplish in colour, the bird does not eat much, and may have a difficulty in swallowing or breathing. In some cases the first symptom noticed is a swelling of one or both eyes, usually one.

The lesions consist of greyish-yellow, cheesy-looking masses situated in the mouth, throat, nostrils, or eyes. When, as is often the case, attention is directed to the disease by a swelling of the eye, pressure by the fingers will cause a fairly hard mass of the deposit to come out from the inside of the lower eyelid. On opening the mouth in such cases similar deposits will usually

be found—in fact, in the majority of cases the disease commences in the mouth as small, greyish, discrete spots which will be found in some of the in-contact birds. These spots increase in size and run together, and attain a size that nearly occludes the larynx, making it most difficult for the bird to breathe even with its mouth wide open.

The course of the disease is progressive rather than rapid, and lasts from one to a few weeks before a fatal ending takes place. The disease is more fatal to young birds; old birds appear to possess a greater amount of either natural or acquired immunity.

Treatment.—Treatment consists of removing the diphtheritic deposit and painting the raw surface with a solution of corrosive sublimate 1 to 1,000 or less, or 2½ per cent. solution of nitrate of silver once or twice daily.

But in view of the fact that apparently recovered birds are liable to suffer from relapses, and even if they do not they may act as carriers of the infection, and, further, that birds during treatment act as agents in spreading and keeping the disease alive, perhaps the best measures from an economical point of view are those of eradication. These may consist of one or two plans. The first is, after a thorough examination, to remove all the apparently healthy birds to non-infected quarters, afterwards examining each one every day, isolating all suspicious birds, and destroying and burning all infected ones.

The second plan is to make a complete clearance of the poultry by killing all the healthy birds for the market. The plan adopted will depend upon the circumstances prevailing in each case, but whichever is taken, the infected quarters should be thoroughly disinfected and kept free of poultry for three months.

CANINE DISTEMPER

Synonyms.—This disease has been given a great variety of names, but now in the English language it is usually referred to as above or as distemper of the dog, and by various names in other languages.

History.—Some doubt exists as to the length of time the disease has been recognised. In Europe it is supposed to have occurred since the second half of the eighteenth century, having been introduced from Asia or Peru. Although the ancients have not described any disease of the dog that can be recognised as distemper, it is quite possible the disease has existed from very early times. It is said to have decimated the canine race in Bohemia in A.D. 1028. At the present time the disease is nearly world-wide in occurrence.

Nature.—Distemper is a prostrating, contagious, and inoculable disease affecting the different varieties of the domesticated dog; the ferret may contract it, and it is generally asserted that several wild species of the dog tribe such as the fox and jackal are subject to the disease. Clinical experience appears to show that the cat is immune to the disease of the dog, neither does the dog contract the distemper that cats suffer from, although it has been claimed that their diseases can be transmitted from one animal to the other by experimental inoculation.

The infectious nature of the disease was proved in the middle of the last century by several experimenters transmitting the disease by means of nasal discharge, saliva, and skin eruption from affected to susceptible young dogs.

Various organisms found in the disease by the earliest investigators were each in turn attributed as the specific cause of distemper. In 1905 the experiments of Carre led him to the belief that the disease was caused by a filtrable virus, and this belief was accepted by many; but in 1910 to 1911 Ferry, McGowan, and Torrey found a cultivatable bacterium, the *Bacillus bronchisepticus*, which they regard as the cause of the disease; therefore at the present time some doubt exists regarding the causal organism, and further results of investigations are awaited before coming to a definite opinion on the point.

The disease usually affects dogs in the first year of their lives; it is the exception for puppies whilst still suckling or for dogs over three years old to contract the disease, but cases do occur at both these stages. Recovery from an attack of the disease usually apparently confers lifelong immunity, but this may perhaps depend to a great extent upon the dog being able to come frequently in contact with the disease and so renew his resistance to it. Certainly dogs do suffer from recurrences of the disease, which may be accounted for by the dog having been removed from all source of infection for some time, and so lost its immunity, or by the animal possessing a great susceptibility to the disease, or by a different strain of the virus, or, as occurs in some other diseases, through some lowering influence the animal is less able to resist the disease.

At times it appears as if certain classes of dogs have a much greater natural resistance or immunity to the disease than others. No doubt the law of the survival of the fittest holds here, and after distemper has raged for a few years the dogs that have survived it and their progeny have acquired a certain degree of natural resistance. Some breeds of dogs appear to possess greater resistance than others; this to a great extent depends upon how they are bred and the life they lead. In-breeding and close breeding are predisposing causes, and so is keeping a number of dogs together, such as a pack of hounds; and if the shepherd's dog and the farmer's mongrel were subjected to similar conditions, they would probably develop the disease in a more severe form than they usually suffer from.

A characteristic of distemper is the different forms it may assume, or perhaps it would be correct to say that in certain years or outbreaks one symptom is pronounced and more frequently present than others. It may be the skin eruption, or the nasal form of the disease, or the abdominal form attended with diarrhœa, or the nervous form of the disease. All these may be met with in the same outbreak, yet one form will be much more predominant one year and another the next, perhaps.

The contagion of the disease exists in the discharges and excretions from the body and in the blood, but experiments apparently prove that it exists to a very limited extent in the later stages of the disease. The natural mode of infection appears to be by direct contact; experiments apparently prove that the disease is not conveyed by the air. The principal source of contagion is the congregation of dogs as at dog-shows, kennels, dealers' yards, etc.

Some authorities are of the opinion that the infection may remain latent in a dog's system for some considerable time before it breaks out, and that this may account for what appear to be spontaneous outbreaks of the disease. In connection with this matter is the question of a certain proportion of the recovered animals acting as " carriers " of the disease for a lengthy period after having recovered from it, so from clinical observation alone it is frequently difficult, if not impossible, to determine by which means such an outbreak may have occurred.

Symptoms.—The first symptoms of the disease consist as a rule of dulness, slight elevation of temperature, the animal may be off its feed, and diarrhœa or vomiting may be present. These symptoms are frequently overlooked, or, if seen, may be attributed to some other cause, such as the animal eating something that has disagreed with it, and it is not until more pronounced symptoms have appeared that the disease is recognised.

The symptoms of the disease may be manifested in several forms; usually more than one is present in each case. The skin symptom is a fairly constant one, and consists of a vesicular eruption on the hairless parts of the body—the abdomen, inside the thighs, etc.—that greatly resembles the poxes in other animals, and was for some time believed to be " dog pox." A veterinarian, on examining a dog suspected to be suffering from distemper, usually early in his examination searches this part of the body for the rash, the vesicles, or their remains, to assist in his diagnosis.

One of the earliest of the pronounced symptoms in the dog is a cough that is frequently and aptly described as if the " animal had something in its throat," which is very near the truth, as the cough is an effort to dislodge some mucous or muco-purulent material. This is sometimes coughed right out upon the ground, at others only into the mouth and swallowed. This cough is very distinctive, later on it becomes husky and more frequent, the animal every few minutes emitting two or three husky, rasping, small coughs; still later the cough becomes moister. This form of the disease, when severe, is liable to be followed by bronchitis or pneumonia, which are serious complications that often end fatally.

Another pronounced symptom is the nasal form of the disease, which consists at first of a watery discharge from the nostrils or the eyes, which later on becomes thicker, and finally it may become very much like pus; it frequently is very sticky and glues the eyelids together, particularly in puppies. In this form of the disease the animal usually sneezes frequently to dislodge this

discharge, which may be tinged with blood from the nostrils, and rubs the nose with the front paws or along the ground for the same purpose. The inflammation of the mucous membranes of the eyelids frequently extends to the cornea (front of the eyeball), and results in ulceration, which may lead to the loss of the eye, but frequently leaves nothing worse than a small scar or no permanent mark at all behind.

Another form of the disease is that affecting the alimentary tract, which is manifested usually in a persistent diarrhœa, which may be accompanied with a considerable amount of straining and occasionally the eversion of the back bowel (rectum). Provided the diarrhœa is not accompanied by marked emaciation, that it does not cease abruptly, and that the fæces do not become very fœtid, the symptoms need not be regarded as really serious.

The nervous form of the disease or nervous symptoms are usually serious; they may be exhibited early in the disease in the form of twitchings of a set of muscles such as those of the eyes, ears, or mouth, or of the whole body, as in an epileptic fit, and end in death. Usually they do not come on until later on in the disease. They frequently follow a severe attack, but they may also develop after a very mild form of the disease has been apparently successfully passed through. Especially is this so with chorea or St. Vitus' dance, which is a common sequel to distemper and affects one or more sets of muscles, those of the head and fore limbs being perhaps the commonest affected. Chorea may disappear entirely, the movements becoming less severe as the animal gathers strength and tone, but the twitchings frequently persist throughout life, or they may lead to paralysis, which is another sequel to distemper; but it is not necessary for it to be preceded by chorea. In a number of cases paralysis of two or the whole four of the limbs may appear independently.

The form of the disease attended with diarrhœa is frequently seen in puppies that are still suckling, when such young animals contract it from a mother suffering from the disease. Vomiting as well as diarrhœa is commonly present, and worms may be vomited or passed in the stools as a coincidence, and a wrong diagnosis and treatment may be attended with fatal results.

In South Africa, until a few years ago, distemper in dogs and biliary fever were confused to a great extent, and the biliary form of distemper was frequently spoken of. Probably in a small number of cases of pure distemper jaundice may occur, but in the great majority of cases in South Africa, when it occurs, it is due to distemper being complicated with biliary fever, and the reports

of cases of distemper having been cured by the trypan-blue treatment in all probability have been either such complicated cases or just pure cases of biliary fever.

The duration of the disease in mild cases may not be more than a week or two, but it usually takes a month or five weeks to run its course; complicated cases take longer.

Treatment—*Preventive.*—A number of dog-owners seem to be fatalists by choice so far as distemper is concerned, and believe if a dog is to get distemper, he will get it, and that ends the matter. Of course, there is no more reason why a dog should contract distemper than any other animal should become infected with contagious disease, or that a child should contract any one of the various contagious infantile diseases. In all it is the same: before the disease can be contracted contact must be made with the contagium, and if dogs are prevented from doing so they will not develop distemper. Under ordinary circumstances, owing to the disease being so widespread and frequently so prevalent, it is very difficult of accomplishment; but owners of kennels can and do keep their animals free of the disease by recognising the above principle and giving effect to it by means of quarantining all fresh animals or those in the kennel that may come in contact with the disease at dog-shows, when visiting stud dogs, etc., for a month before allowing them to mix with the rest of the kennel, and by the employment of disinfection, which readily destroys the contagium.

Another form of preventive treatment is by immunisation. Vaccination was the first method tried. When this was done with the discharge from an affected dog, it was found that a virulent form of the disease frequently resulted, therefore the method found no favour and was discontinued. Cowpox and smallpox vaccination was also tried, with no favourable results accruing.

The next step in immunisation was by the employment of pure cultures of organisms. A number of investigators endeavoured to confer immunity to the disease by one or other of the different organisms found in the disease, each in its turn believed to be the actual cause of it.

Then followed the trial of polyvalent vaccines, which in turn were followed by antidistemper sera, obtained by inoculating horses, and which it was hoped would have curative as well as preventive properties.

At the present time there are various antidistemper sera on the market, and although great claims have been made in favour of

them by their inventors, which claims have been at times supported by others, the weight of opinion at the present time is that a method of bringing about artificial immunity has yet to be discovered.

Medicinal.—The animal should be suitably housed in a place that is as nearly as possible of equal temperature day and night, free of draughts and dampness. The bedding, whatever it is composed of, must be kept clean and free from the discharges and excreta of the sick animal; the quarters should be kept free from offensive odours by the use of disinfectants. The body temperature of the animal can be regulated by a coat (dog), a rug, or even a sack being put upon the animal. Whilst voluntary exercise should not be debarred, only a very moderate amount should be allowed, and forced exercise, such as the animal following its master, should be prohibited until it has thoroughly recovered.

Next in importance to the hygienic treatment of the patient comes its food; the nature of this will, of course, depend upon the severity of the disease. In mild cases it may not be necessary to alter the patient's diet to any great extent, but, broadly speaking, the food should be of such a nature that it is easily digested and sustaining; this is of great importance in severe forms of the disease, or even in milder forms where delicate dogs are concerned. The best foods are first milk, then raw eggs, beef-tea, chicken or mutton broth, and scraped, minced, or finely chopped raw beef. These foods are the foundation upon which to work, and to them may be added rice and bread. They may be combined in various ways—for instance, rice-pudding, milk, eggs, and rice. Even if the appetite is good, the animal should not be allowed to overload the stomach at one time, although it is most desirable to keep the patient's strength up in all cases. Frequently the appetite is very poor, and in some cases entirely lost. If this is so, it is advisable to forcibly feed the patient little and often with one or more of the foregoing foods, to which small quantities of brandy may be added, with benefit to the patient.

Although it will appear strange to many, the actual medicinal treatment of distemper is not of such importance as the hygienic and dietetic treatment of the disease. Distemper, being a specific disease, will run its course despite what may be done to stop it, and it is by careful nursing and feeding from the onset that complications are frequently avoided. Provided the patient is progressing favourably, the less medicinal treatment given the better, and heroic treatment should never be resorted to. One of the

best drugs for distemper is quinine, which may be given in ½ grain up to 5 grain doses twice daily, according to the size of the patient. If the patient is inclined to constipation, ¼ grain up to 2 grains of calomel should be given, and if necessary a warm-water enema with a little glycerine added may be used to empty the back bowel.

If diarrhœa is present, this should be controlled by repeated doses of subnitrate of bismuth in 5 grain doses, or tannoform in doses of from 10 to 50 grains thrice daily; and if vomiting is present, bismuth carbonate in doses of 5 grains should be given, and all exercise in both cases should be stopped.

For the nervous forms of the disease bromide of potassium in doses of from 5 to 10 grains may be given; but for chorea it may be said that all known treatments are unavailing, and the only hope of recovery lies in building up the animal's health and strength, but very few animals ever lose the muscular twitchings once they have developed.

For the eye and nose form of the disease the parts should be frequently washed clean with lukewarm water and smeared with vaseline. If there is much inflammation in the eyes, a saturated solution of boracic acid should be poured between the eyelids twice daily.

When the lungs become affected, the chest should be painted with tincture of iodine twice and covered with a woollen or flannel jacket, and stimulants given internally.

RABIES

Synonyms.—Canine Madness, Hydrophobia (human subject).

History.—Rabies is one of the oldest known diseases. Aristotle in the fourth century B.C. wrote of it as follows: "Dogs suffer from madness, which puts them in a state of fury, and all the animals that they bite when in this condition become also attacked by rabies." Thus, even at that early period the disease was recognised to be infectious.

Many serious outbreaks of the disease have been recorded at various times in different parts of the world. A notable outbreak in wolves occurred in 1803. It began at the foot of the Jura Alps, and continued to 1835, spreading over nearly the whole of Switzerland.

Outbreaks of the disease have occurred in all the great continents of the world, with the exception of Australia.

In April, 1893, the disease in dogs was diagnosed by Mr. Britton, veterinary practitioner, as existing at Port Elizabeth. The history of the outbreak was to the effect that an Airedale terrier was imported at Port Elizabeth in September, 1892. This animal subsequently to landing exhibited symptoms suspicious of rabies, and died a few weeks after.

The next case was observed in January, 1893, and numerous cases were traced subsequent to that, until the date on which Mr. Britton recognised the disease and reported it.

Energetic measures were immediately taken to eradicate the disease, which is only recorded as having occurred in dogs and cats and in one herd of cattle; and although cases occurred at some distances from Port Elizabeth, the disease was quickly stamped out.

In August, 1902, the disease appeared near Bulawayo, and was diagnosed by the writer. Considerable incredulity existed for some time amongst the European portion of the population as to the nature of the disease, but it was not so with the natives; the old men amongst the Matabele stated that the disease was prevalent in the country in their younger days, but eventually disappeared. The natives living in the vicinity of Umtali like-

wise confirmed a previous existence of the disease in that part of the territory.

There is but little doubt that the disease was introduced into Southern Rhodesia from some place north of the Zambesi River. At that time communication and traffic by road was opening up to a considerable extent between Southern and North-Western Rhodesia, and according to reports of travellers the disease was present to a great extent in the latter territory. It was stated that King Lewanika of the Barotse tribe, before his departure for England to attend the coronation of His Majesty King Edward VII., gave instructions that owing to the existence of rabies all the dogs belonging to his people were to be killed. This may sound rather a drastic measure to take, but to eradicate rabies in any country requires drastic measures; and in uncivilised countries the task may appear absolutely hopeless, except by what may amount to the practical total annihilation of the dog population, and even when that is done the wild carnivora are left to carry on the disease.

In Southern Rhodesia the problem presented by the outbreak of the disease was a most difficult one. At that time there were no restrictions on the number of dogs in the shape of a dog tax or in any other way, consequently the country was full of all descriptions of dogs, amongst which were an innumerable number of ownerless curs that roamed town and country as they willed. To rid the land of these unfortunate and unwelcome animals the police were provided with shot-guns, after the necessary authority to deal with the disease had been obtained and regulations promulgated for doing so. On November 22, 1902, less than three months after the recognition of the disease, the police reported the destruction in Bulawayo and the surrounding districts of 9,483 dogs, of which thirty-two were visibly affected with the disease, and that fourteen persons and in two instances "several" persons had been bitten. The "several" referred to natives, where it was impossible to obtain reliable information as to numbers.

By the end of March, 1903, the number of stray dogs destroyed was 29,500 in Matabeleland and 8,759 in Mashonaland. Up to the end of March, 1904, the number of dogs put to death for non-compliance with the muzzling regulations totalled 60,000. A great number of dogs were also killed by civilians and natives that were not recorded. It is impossible to estimate the total number destroyed, but the foregoing figures give an idea of the nature of the task of eradicating the disease.

In September, 1902, the Administration of Southern Rhodesia arranged for Dr. A. Loir, M.D., of the Pasteur Institute, Paris, to proceed to Rhodesia, where he arrived early in October, and immediately upon arrival commenced treatment, with material that he had brought from Europe, of five persons who had been bitten by rabid animals. At the same time Dr. Loir commenced the establishment of a Pasteur Institute locally, where the necessary material (brain) for inoculation could be prepared and people treated, also the training of medical and veterinary assistants to conduct the institute after his departure.

With regard to diagnosis of the disease, amongst the earliest cases of rabies that occurred was that of a dog upon which a post-mortem examination was held by Dr. Hutcheon, Cape Town; Dr. W. Robertson, Grahamstown, both of whom happened to be in Bulawayo at the time; Mr. C. E. Gray, at that time in the Rhodesian Service, now Principal Veterinary Officer of the Union; and the writer. From the history of the case and the post-mortem appearances, particularly the stomach contents, our united opinion was that it was a case of rabies. This dog had bitten a horse on the nose, which latter animal the writer took to his stable for observation. By the twenty-sixth day the animal developed furious rabies, and was destroyed for humane reasons and because it became dangerous to approach. From the brain of the horse the writer and Dr. W. G. Clark experimentally inoculated three rabbits. Three weeks after inoculation, on October 20, one of these rabbits showing the typical paralytic symptoms of rabies was handed to Dr. Loir; from this animal and the history of the sequence of cases Dr. Loir was enabled to arrive at a decision confirming without any further doubt the existence of rabies. The disease radiated out from Bulawayo in all directions. The Transvaal Government took all possible precautions to prevent the disease travelling south, including the clearing of all its canine population from a belt of country along the Rhodesian border fifty miles wide, and also taking other preventive measures. The disease did not cross into the Transvaal, and the other States in South Africa owe a debt of gratitude to the Transvaal for the precautions taken.

The trend of the disease was more noticeable in an easterly direction, and in the course of some months it reached the eastern border at Umtali.

As the dog population decreased in number and became under better control, so the incidence of the disease likewise decreased; but recrudescences and single cases continued to occur until

August, 1913, when the last known case of the disease in Southern Rhodesia occurred in Salisbury. Therefore the disease existed in the territory exactly eleven years.

Nature.—Rabies is an acute, specific, infectious, and inoculable disease affecting principally the nervous system.

It is probable that every warm-blooded animal is susceptible to the disease. In the outbreak in Rhodesia every species of the domesticated animals became infected to a greater or less extent. It is impossible to say to what degree the wild animals were affected, but there is every reason to believe that the wild carnivora were infected to a considerable extent. Several animals were reported as having been bitten by jackals, and in one instance a jackal apparently suffering from rabies passed through the streets of a township. Judging from the apparent diminution in numbers, the wild dog (*Lycaon pictus venaticus*) must have suffered heavy losses, and it is only during the last few years that they are again beginning to make their presence felt as destroyers of domestic stock.

Rabies has long been recognised as a specific disease due to infection alone, and at the present day it would seem to be unnecessary to point out that there is no such thing as spontaneous rabies; but it is not many years ago when a number of people could be found who believed that, under unfavourable conditions, particularly heat and thirst, dogs were liable to become rabid. There is no condition or ill-treatment such as heat, cold, thirst, or hunger, etc., that will ever cause the disease.

Claims of the discovery of the causal organism of the disease have been made by a number of observers at different times, but conclusive proof has always been lacking. In 1903 Remlinger showed that the virus of rabies was filtrable, that it failed to pass through the Chamberland close-grained B and F filter, but that under certain conditions it passed through the more open textured Berkefeld filter.

Remlinger's investigations have since been confirmed by other observers, and the conclusion has been arrived at that the " virus " of rabies is beyond the present power of the microscope. The disease is therefore included in the group of diseases caused by ultra-visible viruses.

In connection herewith mention may be made of what are known as " Negri bodies." In 1903 Negri discovered in many nerve cells obtained from rabid animals corpuscles having special staining properties. These corpuscles can be found in all parts

of the nervous system, but are more numerous and constant in the large pyramidal cells of the hippocampus.

The presence of these corpuscles affords a rapid method of microscopical diagnosis, but they are not present in all cases of the disease; therefore it follows that their absence should be taken as non-conclusive, but their presence may be held to be positive evidence of the existence of rabies.

The only absolutely certain method of diagnosis is by experimental inoculation, the rabbit being the animal usually selected. The procedure is as follows: A piece of the spinal cord, that portion within the first one or two neck bones adjoining the brain, is taken for preference. It is essential that this should be fresh; if the animal has been dead for some time, there is the danger of it having become contaminated with other organisms, in which case the inoculated rabbits may die from a local inflammation of the brain caused by these organisms before the diagnostic symptoms of rabies can appear.

This portion of the cord is rubbed down in a pestle and mortar with sterilised water, and a small quantity ($\frac{1}{2}$ c.c.) of it is inoculated by means of a hypodermic syringe under the covering of the brain (dura mater), and to avoid mishaps not less than three rabbits are employed. The operation is not altogether quite so delicate as at first sight it appears to be. A special trephine is necessary to remove a disc of bone over the brain; the dura mater is then picked up and slightly stretched, the needle inserted and the solution injected.

If the operation has been aseptically performed, and the case is one of rabies, any time after fourteen or fifteen days, which is the period of incubation, the symptoms of rabies will appear; these consist of gradual paralysis, commencing in the hind limbs. The test is to lift the rabbit up by the ears and allow the hind legs to rest on the edge of a table. A healthy rabbit will invariably use its hind legs in this position to push its body forward. The inability to do this is the first sign of the approach of the paralysis, which rapidly extends forward in the body, and in two or three days the rabbit dies. During the time it is sick it does not show any brain symptoms or desire to bite.

In the first experimental inoculations mentioned, owing to the lack of a suitable trephine, the solution was injected direct into the eyeball; afterwards the brain inoculation was performed.

The period of incubation varies within wide limits. In Rhodesia a great many cases in dogs occurred about three weeks after the animals had been bitten; many cases were also reported

from farms, in which the history was that a dog suspected of suffering from rabies had been seen at or crossing a farm, or that there had been a commotion of dogs fighting during the night about six weeks previously to the outbreak of the disease. Probably these periods may be taken as the incubation of the disease in the majority of cases. Authentic cases are on record in other lands of the period of incubation in the case of the dog and man running into several months.

Cases have also been recorded where a period of one or more years is supposed to have elapsed; but without very conclusive evidence, which it is held has not been produced, the accuracy of such long periods of incubation is accepted with great reserve. The matter is one of considerable importance, particularly to the mental attitude of a person who has been bitten by a rabid animal, even if such a person has gone through a course of protective treatment. Six months is the period that experience teaches; if successfully passed, little further fear of the development of the disease need be apprehended.

Statistics of very many thousands of cases of people who have been bitten by undoubtedly rabid animals show that after a course of protective vaccination less than 1 per cent. of deaths subsequently occurs.

The site of the bite considerably influences the mortality; the face and hands are particularly unfavourable. This, perhaps, is influenced greatly by those parts of the body not being covered with clothes, which tend to arrest the poison of the disease being inoculated by the teeth; but even in people not subjected to protective vaccination, the mortality over several thousand recorded cases is only about 15 per cent.

The method of infection is almost without exception through the bite of a rabid animal, but cases can and have occurred through a rabid dog licking a wound or abraided surface on the human body; and a case was recorded of a woman whose dress had been torn by the bite of a dog, and who, on sewing it up soon after, accidentally pricked her finger with the needle, and so inoculated a fatal infection of rabies.

The virus is present in the saliva of dogs, and the length of time it is present before the animal shows visible symptoms of the disease is a matter of great importance, if a difficult one to settle. Cases have been reported of infection resulting from the bite of a rabid dog several days before the animal showed symptoms of the disease. It has been experimentally shown that the virus may be present in the saliva at least two days before

clinical symptoms manifest themselves, and it has been advised that a person bitten as many as eight days before signs of the disease are shown should undergo protective vaccination.

The virus has been found in the milk of affected animals and in the blood, but usually it is entirely confined to the nervous system and the saliva.

Symptoms.—Rabies being a disease affecting the nervous system, it follows that the principal symptoms are of a nervous character, which may take the form of depression or excitability; frequently the former is followed by the latter, excitability being predominant and present in the great majority of cases.

Perhaps the most constant symptom of the disease is the desire to bite, which is present in all species of animals that in a natural state adopt biting as one of the means of offence or defence. An increased flow of saliva also appears to be fairly constant. Exaltation of the sexual instincts is in frequent evidence in rabies. The symptoms are manifested during paroxysms of longer or shorter duration, with more or less lucid intervals intervening.

In Man.

It hardly comes within the province of this book to treat with the disease as it presents itself in human beings, but in man the synonym hydrophobia (fear of water) is used, which, so far as the lower animals are concerned, is a misnomer, and such is also frequently the case in the human subject. Two cases bearing upon this point were brought to my notice; in one, that of a young native (Matabele) girl who, when being taken by her parents to seek advice, had to cross a stream of water, great difficulty was experienced in getting her across. Once across, she calmed down and became fairly rational, but on the return journey, a few hours later, her fear and excitement was so great that she died on the bank of the stream.

The other case was that of a European (man) who had contracted the disease by being bitten by his tame baboon. In this case there was not the least fear of water exhibited, but the sight of a whisky-and-soda at once caused spasms in the throat of the patient and a flow of ropy saliva into the mouth. In this case, although the desire to bite was not present, the desire to infect was, which was exhibited by attempts to abraid the skin of the attendants with the finger-nails, and then to smear a handful of saliva over the place.

In Dogs.

The disease in dogs is usually described as existing in two or more forms—furious rabies and dumb rabies. These terms are useful as descriptive of the symptoms presented, but the premonitory symptoms are the same in both forms of the disease.

The first noticeable symptom is a change in the disposition of the dog; this frequently takes the form of depression, with a tendency to hide away in corners and under beds. In other cases the animal becomes more affectionate, manifested by a desire to lick its owner's hands, or by other alterations in its usual habits. It is possible in such cases that the preliminary depression has been overlooked. Following the depression in fairly quick succession come the symptoms of nervous excitability. These at first usually take the form of restlessness: the animal constantly gets up and lies down again; nervousness is frequently present, noises causing the animal to start up. Dogs may occasionally in this stage be seen trying to catch imaginary flies. These symptoms are followed by one of ill-temper, and when this occurs, if the animal is at liberty, one of three things is likely to occur: it may set out on a journey, it may go in search of dogs to bite in the near vicinity, or the case may develop into one of dumb rabies.

In many instances rabid dogs will set out on an aimless journey, travelling many miles. In one instance a dog well known and easily distinguished travelled a distance of forty miles away from and back again to his home, and left a trail of the disease behind him. When on a journey of this sort the dog travels at a steady lope, but only bites at any and all living things that crosses its path. In the other case the animal sets out apparently with a fixed aggressive intention, and will enter the kennels of other dogs to get at them. Very many dogs were reported as being bitten in their own kennels. Healthy dogs apparently know when a rabid animal is about, as often a howl will go up in the neighbourhood, and they will go into their kennels to get away from the dog, which may perhaps account for the number bitten in their kennels.

In " dumb " rabies a paralysis of the muscles of the jaw takes place, which causes the lower jaw to drop and hang down and the saliva to dribble from the mouth. This form of the disease is frequently mistaken for the dog having a bone in the throat, but in the latter case the animal every few minutes will attempt to dislodge the bone with its paws by scratching at the mouth with its front feet; in rabies this does not occur.

FIG. 6.—DOG SUFFERING FROM "DUMB" RABIES, SHOWING THE CONDITION KNOWN AS "DROP-JAW."

Many cases of dumb rabies appear to go direct from the primary stage of depression to paralysis without any intervening period of excitement, and in such cases only dulness and no desire to bite may be exhibited. On the other hand, I have seen dogs with paralysis of the lower jaw in a great state of excitement, and attempting, although unable to do so, to bite various objects.

Paralysis to a great extent apparently not only governs the symptoms presented, but also the duration of the disease; in some few cases paralysis is the predominant symptom from the beginning to the end of a case.

With regard to water, rabid dogs in many instances show a great desire to drink. An animal may be seen to lap at a bowl of water for some minutes, yet at the end of the time the water has not appreciably decreased in volume, probably due to the fact that the muscles of the throat have become paralysed; and it is for the same reason that a change in the voice is observed. A rabid dog in the majority of, if not in all, instances cannot bark properly. At first the noise made is something between a howl and a bark; later on, in the aggressive stage, the animal is voiceless and the attacks are made in silence.

The appetite is usually suppressed. Some animals will continue to take quantities of food; in many cases the appetite is depraved, and the animal will swallow a quantity of foreign substances. In some cases rabid dogs will bite their own bodies, particularly the legs; this is probably due to irritation at the site of the original wound. It is frequently difficult to discover the site of infection, but when it can be done it is found in most cases that great irritation apparently exists there. There appears to be a tendency with rabid dogs to attack the head of the victim, and probably many of the original wounds of infection are in that region.

In many cases of rabies in the dog the animals exhibit a well-marked "squint"; this often occurs fairly early in the disease, before the animal becomes really aggressive or paralysis of the lower jaw takes place. The animal sits very attentive and on the alert to all that is going on around, with glistening eyes, the before-mentioned squint, and an expression that is very suggestive of the disease.

The duration of the disease is variable; about two days may be considered the average period when the animal has become unmistakably rabid, but I have seen dogs live only a few hours after the furious symptoms have set in. On the other hand, animals may live for a week, and probably some animals recover.

Clinical evidence leads to that belief, and recent observations of experimental cases appear to prove conclusively that recovery does occur in rare instances.

Post-mortem Appearances.—The stomach is the only organ that as a rule lends any aid to diagnosis. In dogs that have died of the disease the stomach is usually congested, and in many cases completely empty. In a variable number of instances foreign substances will be found, particularly pieces of sticks and stones.

If the history of the case is known and points to rabies, and the foregoing conditions are found, they afford pretty conclusive evidence, particularly if the disease is known or suspected to be present in the country, that the case is one of rabies.

The absence of food in the dog's stomach, of course, cannot be taken as evidence of the presence of the disease; on the other hand, the presence of a square meal there can very nearly, if it cannot quite, be taken as conclusive evidence that the dog did not die from rabies.

At times the mouth will be found to be injured by the animal in its fury having bitten different hard substances.

In Equines.

Very few cases of rabies in horses occurred in the Rhodesian outbreak, no doubt due to the small number of horses in the country, also to such animals being usually kept in the stable when not working, so that they were always more or less under the protection of man. More cases occurred in mules, but a far greater number in donkeys; these latter animals were very numerous in the country, and were kept under conditions rendering them easily accessible to dogs.

In the Horse.

I take the following symptoms from a report written by me at the time on the before-mentioned horse that was bitten on the nose on August 28, 1902:

"For some time I have thought that the horse was getting nervous and more excitable than usual. On Friday night last—that is, three weeks and one day after being bitten—I noticed that the lip on the bitten side was paralysed and the nose was drawn round to the opposite side.

"Saturday. The lip was not so noticeable, but was slightly flaccid; animal more excitable, constantly grinding teeth, occa-

sionally yawning, then closing mouth with a biting movement; great irritation in bitten spot, constantly rubbing same against wall.

"Sunday. Same symptoms intensified.

"Monday. Animal worse; in a highly excited condition. In addition to the symptoms presented on Sunday, the acts of urination and defæcation were performed with a great muscular effort, similar to the throes of an animal in parturition, and accompanied by a grunt; the irritation of the nose was less, but a spot about the size of a man's open hand on the right shoulder, about 6 inches above the elbow, appeared to irritate the animal; it was constantly biting at this place until it became soaked with saliva. Muscular co-ordination was also upset. The animal would partly lie down, then fall down or over and bite one or other of its hoofs, mostly the off fore, in a most vicious manner, also the floor of the stable. During the night he was constantly biting a zinc bucket that we were unable to get out of the stable, throwing it everywhere until it was battered almost out of recognition. He left the spot on shoulder and started on his breast, and bit this so severely that the blood oozed out.

"Tuesday. Early in the morning he was biting the skin of his breast and holding it in his teeth, at the same time rising up on his hind legs and striking out with his front. He was most dangerous, and would rush with open mouth at anyone and kick most furiously.

"I shot the poor brute through the heart, and made a post-mortem examination. Everything was normal. . . . Dr. Clark and I inoculated three rabbits with a portion of the medulla. . . . The disease in the first stage was more like megrims than anything else. The horse took food up to the last two days, and drank water all the time, but slowly, with several rests."

The foregoing describes the symptoms of a typical case of furious rabies in the horse. Cases are said to occur in which the paralytic symptoms are more pronounced, similar to those in cases of dumb rabies in the dog.

In conclusion, I would advise anyone who keeps a horse bitten by a rabid animal alive for observation or other reasons to have the loose-box strongly built and the door fastened with iron bars, and even then to keep a shot-gun near at hand for emergencies.

In the Mule.

In the mule the symptoms are very similar to those in the horse, but in a good number of cases reported, amongst the first symptoms noticed was that when working in a span the affected animal would pick up the swingle of the mule in front of

it in its mouth, bite it, and perhaps carry it in the mouth all the time until it came to the end of the trek, when, of course, the animal was tied up separately, perhaps watched for a time, and then shot.

In connection with mules, I had a case that I thought was one of recovery, but there was no history of the animal having been bitten and no way of proving the existence of the disease, so the diagnosis is a problematical one. The following is extracted from my report on the case:

"*December* 1, 1902.—Subject, mare mule. Animal showed all the symptoms presented by the before-mentioned horse in the first stage of the disease—viz., itchiness of the extremities, brain symptoms of excitement, shaking of the head similar to megrims, and swaying of the head from side to side for from five to fifteen minutes at a time, similar to a horse known as a 'weaver,' so much that she knocked the skin off above the eyes. During this time the eyes had a wild and excited look in them. Suddenly the animal would recover and soon appear quite natural. She was frequently urinating and appearing as if in season, and neighing very frequently. After a time she could not neigh properly.

"I felt certain she would develop the violent symptoms of the disease, but, strange to say, she recovered and became quite normal."

Unfortunately, the report does not indicate how long the animal presented these symptoms, but so far as I can remember it was two or three days. Farther on in the report I state:

"Dr. Loir saw the mule daily, and, like myself, said: 'Well, if it is not rabies, what can it be?' He tells me cases of rabies have been known to recover, and I certainly think this is one of them."

In the Donkey.

Several cases of rabies occurred in donkeys, and it was amongst this species that I observed in the most marked manner a character of rabies that is present to a greater or less extent in all species of animals affected with the disease, and that is paroxysms of the distinguishing symptoms of the disease. The period of time between these paroxysms is longer in the earlier stages, gradually decreasing as the disease increases in intensity, until in the final stages no intervals are observed; likewise the intensity of the symptoms and the length of time each paroxysm lasts increases with the duration of the disease to a fatal issue.

In donkeys the earlier symptoms shown in such paroxysms may be that the ears begin to move forward and backward quickly, the animal brays, the eyes glisten, and it becomes very alert. These symptoms may only last for the space of a minute or longer, when the animal again becomes apparently dull and uninterested in what is going on around. If the animal is a jack, these symptoms may be easily mistaken for sexual ones, and, in fact, sexual excitability may be manifested. If there is no visible cause for this, suspicion may be excited; in the case of a gelding it would be. Very soon, if not even at the first manifestation of such symptoms, will be added the one of biting at whatever may be within reach of the animal, such as posts if the animal is tied up, or other animals if at large, which symptoms in turn are soon followed by biting at the site of the original wound if it is get-at-able, as well as the legs and extremities.

In Cattle.

Several head of cattle were reported to have died of rabies during the outbreak in Rhodesia. The symptoms are similar to those presented by other animals, except that, so far as my experience goes, attempts at biting are not made by cattle.

In affected animals there is restlessness, with the eyes prominent and staring, giving the animal a wild appearance; a dribbling of saliva from the mouth, nervous excitability, bellowing, muscular tremblings, and paroxysms of aggressiveness, during which the animal will charge animate or inanimate objects, at times with such fury as to break the horns; but in my experience the outstanding symptom of the disease in cattle is the pronounced sexual desire, which is present to a greater or less extent in all species of affected animals.

In 1907, at Christmas, I examined a herd of cattle on the Insiza River, the history of which was that a dog behaving in a strange manner had been seen at the native kraal at which the cattle were kept about six weeks previously. One cow was sick at the time of my visit, presenting the foregoing symptoms, but the marked character of the case was she kept bellowing for the bull. There were several bulls at this kraal, and soon after one bull had served her she would bellow again, and another bull would come along. Five different bulls served this cow in front of me in a very short space of time, and this went on as long as the bulls were able to do so, practically up to the time of the death of the cow. Seven cows died at this kraal, every one of which the native in

charge stated showed a similar sexual appetite, his statement being confirmed by a European farmer residing only a few hundred yards away.

In Sheep and Goats.

A number of these animals died of rabies. The symptoms, including the sexual appetite, are similar to those in cattle. It was reported that sheep bit each other. I cannot vouch for the accuracy of this statement, and am rather inclined to doubt it.

In the Pig.

The symptoms, including biting, are similar in the pig to those shown by other animals. Squeaking is rather a marked character in some cases of nervous excitability and aggressiveness.

In Wild Animals.

Cases of antelope found dead and suspected of having died of rabies were reported, which were probably correct, but no authentic information was obtainable.

In wild carnivora no doubt many cases occurred, but the information obtainable was meagre: a native herd might report a jackal had bitten one of the animals he was herding, and occasionally jackals suspected of suffering from rabies were seen by Europeans. The wild dogs apparently decreased considerably in numbers during the rabies outbreak. No information of the larger carnivora, lions and leopards, was forthcoming.

In the Cat.

Last, but not least, comes the cat, which perhaps may be described best as "bounding ferocity." My first view of one rabid cat was the animal hanging by its claws high up in the window curtain. The interview was not more than a second or two, but I shall not quickly forget the glaring eyes, tail lashing, ready-to-spring appearance. I expected it to land on my head. Probably it would have done if it had had a better jumping-off ground. Cases of cats killing their kittens and other cats in the early stages of the disease were reported.

Treatment.—Medicinal treatment of the disease is not to be thought of.

Protective inoculation of man should be made available in countries where the disease exists, as was done in Rhodesia.

In practice the treatment resolves itself into prevention and eradication.

In prevention we have the example of Australia, which has been able to keep the disease out by rigid inspection, quarantine, etc.; and we have the example of the Transvaal of preventing the entry of the disease by land. The prohibition of the importation of dogs from centres where the disease exists, and the quarantine of dogs from other places, are imposed by most countries free of the disease.

The measures taken for the eradication of the disease will be governed by the conditions prevailing in the particular country. The simplest and most effective way is that advocated by the Matabele: to destroy all the dogs. Of course, such a method—or, in fact, any other method—will meet with opposition, or at least with people who will try to avoid or fail to observe the conditions laid down; but in whatever country it is, the object to be aimed at is to get the dog population under the best possible control and supervision.

As a preliminary control, registration of all dogs and compelling all dogs to wear a distinguishing badge, through which ownership can be traced, is most useful; this is usually done by means of a dog tax, and the fee imposed according to the amount regulates to a great extent the number of dogs kept. In carrying out the provisions of such tax, all unregistered dogs are periodically seized and disposed of by sale or the lethal chamber.

On the outbreak of rabies various methods of control were adopted in Rhodesia, but the one that had the greatest effect in checking the disease was that any dog at large might be destroyed by anyone, and all loose dogs seen by the police were destroyed. Owing to representations, dogs (sporting) accompanying a person with a loaded gun were allowed to be at large, the interpretation of which is that the dog had to be kept on a chain or lead until the gun was loaded for the sport of shooting. Of course, penalties have to be provided for failure to comply with these or any other regulations.

Muzzling and allowing muzzled dogs to be at large is to a great extent a failure, for several reasons: owners forget to put muzzles on, and deliberately release dogs unmuzzled at night; ineffective muzzles, even approved patterns being allowed to get into disrepair, such as the side straps becoming ineffective; and with the best of muzzles, if a dog develops furious rabies, there is the probability—and it happened in several cases—of the animal so

damaging the muzzle in its efforts to bite that it was enabled to do so eventually.

In conclusion, there is one point I would impress: In the case of a person bitten by a dog during the time that rabies is prevalent, unless the dog shows definite symptoms of the disease, the animal should be isolated, securely fastened up and kept under observation, and if it lives for a week or ten days it is nearly proof-positive that it was not suffering from rabies at the time of biting; the only danger of error creeping in—and it is a very remote one—is that the dog might suffer from the disease and recover. It has been held that dogs never recover, but I have yet to be convinced of the existence of any disease that, when naturally contracted, kills all affected with it.

Through thoughtlessness, as soon as a dog has bitten a person a desire arises to kill the dog. This desire should not be given effect to, as it destroys a valuable source of information and places the bitten person in a disconcerting condition of uncertainty. Whether such a person should immediately undergo protective vaccination will be decided by the circumstances. If there is a doubt about what should be done, it is better to give protection the benefit of it and undergo the treatment.

In forwarding spinal cord for the purpose of diagnostic inoculation of rabbits, perhaps the best tried method which has been found is to cut the dog's head and neck off near the shoulders, pack this in ice if obtainable, otherwise under the best available conditions to prevent putrefaction, and despatch it by the quickest route to the investigator.

Pasteur's Protective Inoculation.—It has been repeatedly proved by experimental inoculation that dogs and other animals can be immunised against rabies by this treatment, and the statistics in mankind are convincing to any unprejudiced person. The method is really a preventive inoculation, but although it can be used for that purpose, it is usually only employed when persons have actually been bitten.

The principle of this treatment is shortly as follows: When a rabbit has been inoculated into the cranial cavity as described, it dies in about fourteen or fifteen days from rabies. By passing the disease from rabbit to rabbit through a number of generations an intensification of the virus occurs, and the rabbits die on the sixth or seventh day after inoculation. When such a stage has been reached, and the virus has been fixed—that is, it will invariably kill rabbits on the sixth or seventh day—it is known as

"virus fixe" of Pasteur, and the spinal cords of such rabbits are the material used in protective inoculation.

Such a cord is aseptically removed from the spine and suspended in a jar, kept at a certain temperature, over fragments of caustic potash, which desiccates (dries) the cord. After being kept fourteen days under these conditions the cord becomes inert, no longer contains living virus, and it is from cords that have been kept a variable number of days that material is taken for the daily injection of the bitten person. In the ordinary method of treatment fifteen injections, one daily, are employed, commencing with cord that has been kept thirteen or fourteen days, and then daily employing cord that has been kept one day less, until cord that has only been desiccated six days is reached; afterwards, the five, four, and three-day cords are used in various orders of sequence, according to the idea of the inoculator, but usually three-day cords are used on the last two days of the treatment.

An intensified method of application of the treatment is adopted at times for reason of the history of the case, the site and extent of the wounds, or the length of time that has intervened between infection and commencement of treatment. In this method a morning and evening injection is used for three or more days, and the employment of the three-day cord is reached about the seventh day of treatment.

PART II

DISEASES CAUSED BY ANIMAL PARASITES

SECTION I

DISEASES CAUSED BY ARACHNIDA

THE Arachnida are air-breathing animals, with the head and thorax usually fused together (cephalo-thorax); the abdomen is often non-segmented, possessing four pairs of legs, but no wings.

They are divided into several orders, only two of which include parasites of warm-blooded vertebrates, and are therefore important to the veterinarian; they are (1) the Linguatulida, (2) the Acarina.

The Linguatulida.—These are somewhat worm-like in shape, and in the adult stage possess no limbs; they are not numerically important.

Linguatula denticulata is the larval form of the parasite that is found in the internal organs of herbivorous animals. By means of dogs eating such organs, the parasite is enabled to reach the nasal chamber of the dog and the adult stage, in which eggs are laid; these are sneezed out by the dog upon grass, etc., and find their way to the stomach of the herbivorous host, and so the life cycle is completed. Whether they exist or have been found in South Africa is unknown to the writer.

The Acarina.—This order is divided into ten families, five of which are important as affecting the domestic animals; these are:

1. *Ixodidæ*, the ticks.
2. *Gamasidæ*, the red mites that chiefly affect rats, rabbits, and fowls.
3. *Trombidiidæ*, the harvest bugs.
4. *Sarcoptidæ*, the mange mites.
5. *Demodicidæ*, the follicular mites.

Ticks.

The ticks belong to the family Ixodidæ. They are of great economic importance by reason of the damage done to animals

through exposure to tick infestation. This can be placed under two headings:

1. That due to the tick itself.
2. That due to diseases transmitted by the tick.

The loss that is brought about by the second of these two is the one that looms most in the public eye, but in all probability the loss due to the former far exceeds that due to the latter, though both represent a very heavy tax on the live-stock industry of the sub-continent.

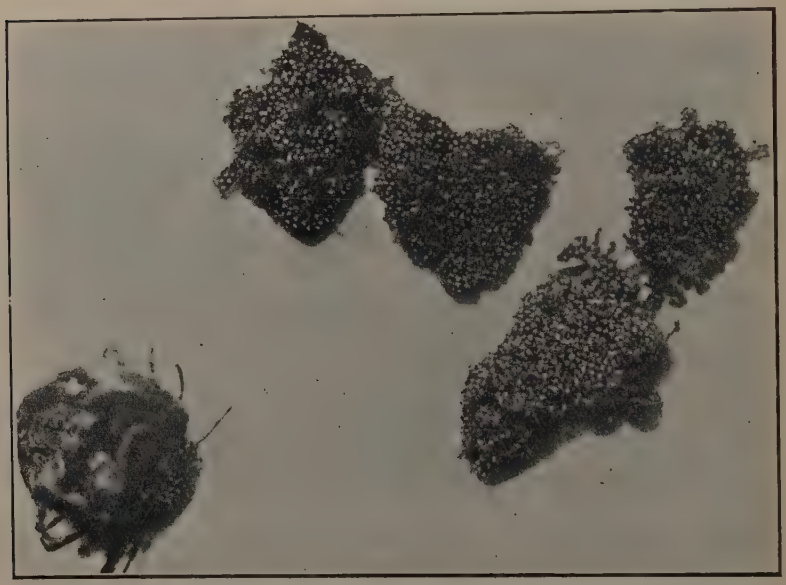


Fig. 7.—Female Tick, showing Clusters of Eggs.

Amongst the losses due to the tick itself are what is known as " tick worry "; this term is self-explanatory, the worry in some cases almost amounting to torture, and prevents a tick-infested animal from growing and thriving as it should do. Then there is " tick poverty," caused by the amount of blood withdrawn by the tick causing anæmia in its host. Experiments were carried out by Sir Arnold Theiler some years ago proving that severe tick infestation can result in the death of the host from acute anæmia. It has been estimated that in ordinary cases of bad tick infestation the ticks remove as much as 20 pounds weight of blood from

mature cattle per month. Needless to say, a large proportion of the food consumed by such animals goes to the making of blood for the ticks, the result being that tick-infested cattle become stunted and slow maturing, a matter of great economic importance. Among the losses due to the actual bite of the tick, mention may be made that such bites open the way for entry of organisms of other diseases and pus-forming bacteria. The

FIG. 8.

1. EGGS LAID BY A SINGLE TICK.
2. THE SAME (CONSIDERABLY ENLARGED).
(Photograph by Mr. R. J. Stordy, Principal Veterinary Officer, British East Africa.)

damage done to the teats and udders of milch cows and heifers render a number of such animals only fit for slaughter purposes. There is also the damage done to the hide; this has been estimated as high as 30 per cent. A great deal of the foot rot in sheep in South Africa is directly due to tick bites. There are other ways in which damage is done, but enough has been said to show how very important the subject is.

With regard to diseases transmitted by the tick, these are dealt with separately, and include redwater, gall-sickness, biliary fever, East Coast fever, heartwater.

Life History of Ticks in General.—The life history is essentially similar in all members of the tick family, but certain species have slightly modified habits.

The engorged female, which is the tick most generally recognised, having completed the process of filling herself with blood, drops off the animal to the ground, and seeks a sheltered spot in which to lay her eggs. Such a spot may be found under a tuft of grass, a stone, a lump of earth, or a crack in the ground. Here in the course of a few days or longer she commences to lay her eggs, the period occupied in the process being from a few days to several weeks, and the result a greater or less number of eggs,

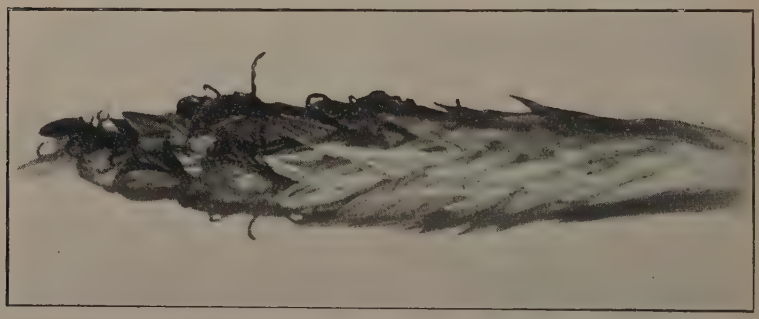


FIG. 9.—TICKS ON GRASS HEAD. (Photo by Bevan.)

according to the species of tick—usually a greater, amounting in some cases to several thousand eggs—and when completed the mother tick has become a dead and empty shell.

Temperature plays a great part in the different stages in tick life, warmth accelerating and cold delaying the time taken in laying the eggs, in hatching them out, and in ticks moulting from one stage to another.

In the course of a few weeks the eggs hatch out and the first stage of tick life is reached, the ticks at this stage being termed "larvæ." These are very minute, and have only three pairs of legs. In the course of a few days they climb to the top of grass leaves and stems, waiting there for some beast to brush past, enabling them to get on to its body and become attached Having accomplished this, they then engorge with blood, and in the course of a few days the majority drop to the ground from

their host and moult, although some species remain on the host to moult, and after having moulted become attached to the same host again. This moulting or changing of the skin varies considerably in the time taken to accomplish; when it is completed the ticks are known as " nymphs," and possess four pair of legs. The nymphs, other than those which moult on their host, go through the same performance of climbing the grass and becoming attached, but the engorgement takes usually a longer time than in the case of the larvæ; then those which do not remain on the same host again drop to the ground, moult, and enter the third or adult stage. In this stage the male is distinguishable from the female, and when they become attached the female engorges enormously; but the male does not, and remains comparatively small, and is usually to be found attached to the skin at the under-surface of his much larger " better half."

Ticks can be, and frequently are, divided into two classes, according to the manner in which the life cycle is completed. The classes are:

1. The continuous feeders.
2. The interrupted feeders.

The blue tick belongs to the first class—that is to say, all the stages from the larva up to the engorged fertilised female are passed on the same individual animal. Many of the ticks are interrupted feeders—that is, they drop off to moult, and spend the succeeding stage on a different animal. A knowledge of these habits and length of time spent at the different stages on the host, and the length of time the tick can exist without a host, is of the utmost importance when considering measures for tick destruction and the eradication of tick-borne diseases.

The following ticks have been recognised in South Africa. Many of them are the transmitters of disease. The blue tick, the brown tick, the bont tick, and the red tick, are the commonest species present, and are the greatest transmitters of disease. The Ixodidæ has been divided into two subfamilies, the Ixodinæ and the Argasinæ.

Ixodinæ.

. *Blue Tick* (*Margaropus decoloratus*) is the greatest transmitter of redwater and gall-sickness in cattle. It is a continuous feeder, and remains on the same animal for about twenty days; consequently it is very easy to eradicate by continuous dipping, and when a dipping campaign is instituted this is the first tick to disappear from the cattle. An important point to remember

about this tick is that the disease passes through the egg. Therefore it can transmit disease in the larval as well as in the other stages of its life.

The eggs number from one to four thousand, and hatch out in from three to six weeks in summer weather. The larva can live without a host for six months. The engorged female usually commences to lay eggs five days after dropping from the animal. The adult can live off the host for six months.

The male is less than $\frac{1}{8}$ inch in length, and is completely covered by a shield. The shield is very light brown or reddish-brown in colour, with two horn-like projections in front, extending on either side of the rostrum. The shield of the male is covered with numerous short hairs.

The female when fully engorged is of a bluish colour, with the shield and legs light reddish-brown. The shape is rather oblong and somewhat constricted near the middle. The body is marked by three parallel grooves, and is sparsely covered with short hairs. The length of the engorged female is about $\frac{3}{8}$ inch. The shield has longer hairs.

The Brown Tick (*Rhipicephalus appendiculatus*) is the principal disseminator of East Coast fever, but it can also spread redwater and gall-sickness; it is an interrupted feeder, and becomes attached to three hosts during its development. The eggs hatch in from five weeks to as many months, and the larva can live seven months without a host. When it becomes attached it engorges quickly, and drops off in from three to eight days; it is then off from sixteen to twenty-one days, and as a nymph it can so live for six months; it is on the second host again from three to eight days; it is then off the host for eighteen days, but can live as an adult without a host for fourteen months. When it becomes attached in this stage it remains on the host four to seven days. The disease does not pass through the egg, but if a larva or nymph feeds on an animal suffering from East Coast fever it can convey the disease to a healthy beast during the next stage of its life—that is, either as a nymph or an adult; but if a larva that has fed on an East Coast fever animal and as a nymph feeds on an animal not subject to the disease, the tick discharges its poison and cannot infect a susceptible beast in the adult stage of its existence. It is of a bluish-brown colour. The male is oval, and twice as broad behind as in front. The shield is dark brown and marked with large and small punctures. The latter are very numerous. The length of the male is about $\frac{1}{8}$ inch. The engorged female, nearly $\frac{1}{2}$ inch in length, is

elliptical in shape. The colour of the female is bluish-brown. The abdomen is marked by three parallel grooves. The integument of the abdomen is very thin, and with a few hairs. The length of the shield is greater than its width, and the eyes are flat. The legs are dark brown.

The Red or Red-legged Tick (*Rhipicephalus evertsi*) is the transmitter of biliary fever, spirochætosis, redwater, gall-sickness, and East Coast fever. It is an interrupted feeder, and has two hosts for its three stages, the larval and nymphal stage being passed on the same host. The eggs, numbering several thousands, hatch in thirty days, and the larva can live seven months without a host. When it becomes attached it is on its first host as a larva and nymph for a period of fifteen days; it then drops off and has a second moult on the ground, and remains off for about twenty-four days. Biliary fever of equines and East Coast fever do not pass through the egg; therefore their transmission depends upon the tick feeding as a larva or nymph upon an animal with the disease in its blood, and transmitting it to a susceptible animal in the adult stage. Spirochætosis, on the other hand, does pass through the egg; therefore the tick can transmit it at any stage of its life.

It is a very common tick; its favourite sites are in the ears and under the tail. It is found on cattle, sheep, horses, mules, donkeys, goats, and many wild animals, also on dogs.

The male is about $\frac{1}{5}$ inch in length. The body and the legs are a deep orange yellow, with the rostrum and shield dark reddish-brown or blackish. The shield is very densely punctured, as in the Cape brown tick. The eyes are placed in orbits. The female may be over $\frac{1}{2}$ inch in length when engorged. The colour is brown, often marked with reddish spots, while the shield is blackish-brown. The legs of the female and also the eyes are as in the male.

The Eyed Tick (*Rhipicephalus oculatus*) is found in the Transvaal and Rhodesia. This tick is similar to the red tick in having the eyes in an orbit, but differs from it in that the legs are reddish-brown rather than orange yellow, and the punctures on the shield are distinct.

The Cape Brown Tick (*Rhipicephalus capensis*) is also similar to the brown tick, but can be distinguished by the shield being finely punctured, and these punctures are so numerous that they run together, causing the surface to appear as though covered with very fine tooth-like projections. The shield of the male is three times as broad posteriorly as at the anterior end. This

tick can transmit East Coast fever, and has been found on dogs and goats.

The Black Pitted Tick (*Rhipicephalus simus*) is readily distinguished by the shield in both male and female being very dark, almost black in colour, and very shiny. The small punctures are almost invisible; the large punctures are not very numerous, but are comparatively large. This tick can transmit East Coast fever, but it usually spends its larval and nymphal existence on some other animal, such as game. It has been found on the bush pig, wild dog, hedgehog, jackal, lion, and goats.

The Shiny Brown Tick (*Rhipicephalus nitens*) is very similar to the brown tick, but the general colour is reddish-yellow and the shields are shiny. This tick can transmit East Coast fever. In addition to domestic animals, it has been found on the wild dog. This and the black pitted tick are found principally in Cape Colony.

The European Brown Tick (*Rhipicephalus sanguineus*) is very similar to the common brown tick. The characters distinguishing these two species are rather technical. The character that the shield in the female is distinctly longer than it is broad will aid in distinguishing it, but it must be remembered that the shield of the female brown tick is also longer than broad, but not so distinctly so. This tick has been found upon the Cape hare, the ferret, hedgehog, lion, reed buck, and dogs.

Rhipicephalus bursa is a brown tick sometimes found in South Africa. It is very similar in appearance to the common brown tick, but can be distinguished by the shield being plainly as broad as long and by the punctures of the shield being all of one size—*i.e.*, all small. It has been also found on dogs.

The Bont-legged Tick (*Hyalomma ægyptium*) is a most voracious feeder, and when on the ground it is constantly on the move, running about quickly looking for a host. It requires two hosts, the larval and nymphal stages being spent on the same animal, mostly on birds, hares, and other wild animals, also on poultry, the favourite site for it being on the head and neck. Adults have been found on various antelope, wild pig, reed buck, ostrich, sheep, goats and dogs, as well as cattle. It attaches itself to the under parts of the body principally, such as the feet, legs, behind the shoulder, around the genital organs, udder, etc.

It is found principally in the driest parts of South Africa, and is most prolific; as many as 13,000 eggs have been laid by one

female. The eggs have great vitality, and can withstand very dry conditions.

Experience has proved that this is one of the most difficult ticks to kill and eradicate by regular dipping. The difficulty in killing it is attributed to its long proboscis, which enables it to penetrate deeply and draw its nutriment below the layer of the skin impregnated by the arsenical dip. The difficulty of eradicating it is due to the fact that it spends only its adult stage upon cattle, but even that stage may be spent upon some other animal.

Its bite causes abscesses and sloughs, particularly in dogs and calves, and foot rot in sheep. Experiments to date have not proved this tick to be the transmitter of disease, but popular opinion, founded to a great extent upon the poisonous character of its bite, is very doubtful about this being the case, and suspects it of transmitting gall-sickness.

The male body is flat and oval, with the posterior end rounded. The length with the rostrum is about $\frac{1}{8}$ inch. The shield is large, covering nearly the whole body. The colour of the shield is brown, closely and finely punctured. This tick can readily be distinguished by the legs being ringed, reddish-brown, and whitish.

The engorged female is $\frac{1}{2}$ to $\frac{3}{4}$ inch in length. The shield is like that of the male, only smaller. The rest of the body is a greyish-bluish brown or brownish, sometimes whitish when very full of blood. The legs of the female are like those of the male. The colouring of the legs ("bont"=speckled) is the best character for determining this species.

The Bont Tick (*Amblyomma hebræum*) is the transmitter of heartwater. It is an intermittent feeder, and requires three hosts. The disease does not pass through the egg, but if contracted in the larval stage it is transmitted by the nymphæ, or if contracted by the nymphæ it is transmitted by the adults. A difference exists here to what pertains in the brown tick and East Coast fever. If a larva feeds on an animal suffering from heartwater, and as a nymph feeds on an animal not susceptible to the disease, the tick does not discharge its poison, but as an adult is able to transmit the disease to its third host if susceptible.

When the engorged female drops to the ground and has sought shelter, she rests from two weeks to two months or longer before commencing to lay eggs. The eggs, numbering from ten to twenty thousand, take from several weeks to several months to hatch. The larva takes from four to nine days to engorge, and

after that several months before it moults. The nymph takes from four to eight days to engorge, and some weeks, sometimes months, before it moults. The adult tick remains on the animal from six to ten days.

The bont tick is found along the southern and eastern coast, seldom reaching the high veld. It has never been found in Rhodesia. This tick attacks the bare places on the body of the host, such as under the shoulder, udder, and round the genitals. The larva and nymphs may be found in the long hair of the sides and the back. Besides attacking cattle, sheep, goats, and horses, it has been found on fowls and dogs, on various antelope, buffalo, wild dog, lion, and ostrich.

The male is a handsome creature. A typical specimen is broadly ovate, $\frac{1}{5}$ inch in length, and almost flat. The back is entirely covered by the shield. This is firm to the touch, exceedingly tough, and is resplendent with bright glistening colours. The prevailing tint is slaty-grey, which is broken into patches by irregular black lines edged with green and a tinge of gold. The whole effect is that of hieroglyphic characters inscribed on a pearly shell. The legs are dark brown with paler bands at the joints. The mouth organ or rostrum is one-third as long as the body.

The female varies from almost dead black through green to chestnut brown in colour at the time it gets on to the animal as an adult. The front part of the back only is occupied by a shield, and to this portion is restricted the gay markings of gold, green, and black, which at once serve to identify it with its mate. The female gorges herself with blood, and as she enlarges becomes distinctly reddish-brown, with a band of red around the hind margin. When fully distended she may measure 1 inch in length, $\frac{3}{4}$ inch in width, and $\frac{1}{2}$ inch in thickness. Small specimens may not attain much over half these dimensions.

The Variegated or Green Tick (*Amblyomma variegatum*).—This tick is commonly found in Central Africa, and is rather rare in South Africa. It is found near the Victoria Falls and Umtali in Southern Rhodesia, where it is accused of causing what is known as senkoba skin disease and a form of ulcerative lymphangitis in equines. Besides attacking the domesticated animals, it has also been found on a variety of wild animals. It is similar to the *A. hebræum* in size and structure. The male is a resplendent green colour tinged with gold; the female is the largest tick found in Rhodesia. It also attacks similar parts of the body, and causes a tumour-like swelling of the skin, the size of a pea or

larger, in the immediate vicinity where it has become attached. A search under such swellings usually reveals a male and an unengorged female; by the time the female has become fully engorged the swelling has usually disappeared.

Tortoise Tick (*Amblyomma marmoreum*).—This tick is much similar in appearance to the *A. hebræum*, and has a wider distribution. The adults are found upon tortoises, but the larvæ and nymphs may be found upon cattle. It is not known to carry any disease.

Lizard Tick (*Aponomma exornatum*) is widely distributed, and occurs normally upon snakes, lizards, monitors, etc., but it has also been found upon dogs. It is a very small tick, and can be distinguished by the shield being marked by metallic green spots, nine on that of the male and three on that of the female. This tick may one day possibly be proved of economic importance, particularly if it is correct, as was recently reported in the *Scientific American*, that the snakes in the New York Zoo infested with this tick were found to be infected with, and by means of the tick capable of transmitting, piroplasmosis.

The Dog Tick (*Hæmaphysalis leachi*) is the transmitter of biliary fever in dogs. As in redwater in cattle, a dog that has recovered from biliary fever has the organisms of the disease in its blood, and is capable of infecting ticks that engorge upon it; also, as in redwater, the disease passes through the egg; but in biliary fever in dogs it also passes through both the larval and nymphal stages of the tick, and the tick is unable to transmit infection until it reaches the adult stage. Owing to this condition in the life of the parasite (piroplasma canis) the dissemination of the disease may be enormous, and cover long distances; for instance, an infected female tick lays several thousand eggs, and several females may fall from a single dog. The tick is an interrupted feeder, the eggs hatch out, and the larvæ become attached to dogs, jackals, and other carnivora for about two days, during which time they may be transported long distances; they drop to the ground, moult, the nymphæ emerge and are picked up in a similar manner, and remain attached for four days before dropping to the ground and moulting into adults, at which stage the disease is transmitted to the dog; so it can be imagined how widely scattered the ticks may have become by the time they reach the adult stage.

It is a very common tick all over South Africa, attacking carnivorous animals. The male is reddish-brown, about $\frac{1}{8}$ inch in length. The upper surface is covered by a shield, which is

finely and evenly punctured. The underneath is light, often yellowish in colour, with a few fine hairs and punctures. The eyes are absent in both male and female. The female engorged is rather oval, of bluish or greyish-brown colour; in length not quite $\frac{1}{2}$ inch, often much less. There are three longitudinal grooves on the dorsal surface near the posterior margin. The shield is rather heart-shaped, light reddish-brown, punctures deep and regularly distributed.

The Russet Tick (*Ixodes pilosus*) does not appear to be very common; it is considered to be the cause of paralysis of sheep in the Cape Colony, particularly in the eastern province. The male is about $\frac{1}{8}$ inch in length. The eyes are not present. The shield is horseshoe-shaped and reddish-brown. The body is very hairy. The anal groove passes around the anus in front of it. The lips are slender and hairy. The female, when unengorged, has a flat body, oval in outline, much wider and rounder behind; colour yellowish-red; long whitish hairs on all surfaces; when engorged resembles a castor-oil bean slightly flattened, and becomes a dark bluish colour. Shield yellowish-brown, lighter than the abdomen when young, glabrous, oval, somewhat pentagonal in outline, as wide or about as wide as long. Eyes also absent in the female. The life history of this tick has not been worked out. It has been found on the bush buck and the leopard, and also attacks cattle.

Ixodes rubicundus is a tick somewhat similar to the preceding species. The colour of both male and female is more of a red or brownish. The shield of the male is rectangular, and longer than broad. Little seems to be known of this tick, which has been found in Cape Colony, and is supposed to transmit disease of sheep.

Argasinæ.

The "Spinose" Ear Tick (*Ornithodoros megnini* or *Argas megnini*) has been known in South Africa since 1912. It is a native of America, where it has been recognised for many years as a pest of cattle, sheep, horses, and other domesticated animals.

It has obtained its name of "spinose" from the fact that in the nymphal stage its body is covered with numerous spines, and is the only tick known in which this occurs. The life history, so far as it is known, is as follows: The adult ticks live in walls, cracks in posts, etc., near the cattle-sheds, where they pair, and a numberof eggs are laid. These hatch out into six-legged larvæ, which, on obtaining a host, find their way to the inside of the

ear and become attached. After about a week the first moult takes place inside the ear. The nymph stage being reached, it takes the nymphæ about three weeks to engorge, at the end of which time they leave the ear and fall to the ground, seek cover, and the second moult takes place, and the adult stage, in which pairing, egg-laying, as described, occurs. It will be noted that with this tick a peculiarity exists in that the adults never feed; the nymphæ store up sufficient nourishment to carry the adults through.

The Tampan or Poultry Tick (*Argas persicus*) is the transmitter of spirochætosis in fowls. The life cycle is as follows: The adult female lays from 20 to 100 eggs, usually in cracks or crevices, in the poultry-house or in the bark of a tree or post; from these a six-legged larva hatches out, which in due course attaches itself to a fowl. After engorging, at about the end of ten days it leaves its host and crawls into a crack, where the first moult takes place, and the first-stage nymph appears. Another moult occurs, and the second stage of the nymph is arrived at, which in due course undergoes a third or final moult, and the adult results. The larval is the only stage in the life cycle in which the tick becomes attached to the fowl in the first and second nymphal, and also in the adult stage. The ticks hide themselves in cracks and crevices, and only come out to feed at night, and then for about only two hours at a time. Adults can live without a host for eighteen months.

Tampan (*human*) (*Ornithodoros moubata*) is the transmitter of relapsing fever in man. It is not really of veterinary importance, only the belief is erroneously held that the same tick (*Argas persicus*) that preys on poultry transmits this disease. The life history and habits of this tick are much the same as those of the fowl tampan, only that it infests human habitations and outspans instead of fowl-houses, and its favourite hosts are human beings. The organism of the disease (relapsing fever or spirochætosis) passes through the egg, but it is not until the first nymphal stage is reached that the tick is able to transmit the disease. Once infected, the tick is able to infect for months. It is even stated that infection will persist over the third generation of the tick after it has imbibed infected blood.

The Bat Tampan (*Argas vespertilionis*).—This tick sometimes attacks man; therefore bats should not be allowed to frequent lofts or roofs of houses for sleeping-places.

Tick-Transmitted Diseases.

When the student is confronted with the proposition of determining simply by clinical observation which of the tick-transmitted diseases the animal is suffering from, considerable difficulty may be experienced in many instances in arriving at a correct diagnosis. In fact, this difficulty is not confined to the tick-transmitted diseases alone, but includes diseases transmitted by other means, such as flies and perhaps mosquitoes, which are thought to transmit horse-sickness.

The difficulty arises simply as an effect following a cause, the cause being that in the majority of cases of this class of disease the blood is the seat of the disease, and the disease causes the destruction to a greater or less extent of the blood; therefore the effect that follows is a train of symptoms due to the destruction of the blood that greatly resembles each other in all the different diseases of this nature.

The diseases coming under the heading of Plasmoses are caused by small parasites belonging to the animal kingdom which live on or within the red corpuscles of the blood. These parasites produce poison which affects and generally destroys the corpuscles. In some of the diseases the corpuscles are destroyed to a very great extent, being decreased to as much as one-fifth.

The loss and breaking down of this important constituent of the blood is responsible for the symptoms observed during life, and for the appearances found on post-mortem. One of the functions of the liver is to break up the colouring matter of the blood set free by the destruction of the red corpuscles and convert this into bile; but in these diseases an over-production of bile occurs. This cannot be readily discharged in the ordinary manner; hence reabsorption into the blood follows, and on being carried by the blood-stream all over the body, stains the tissues yellow. The liver, being overworked, throws more than should be upon the spleen; consequently upon post-mortem this organ is found considerably enlarged in some of these diseases. When the dissolution of the red blood-corpuscles has taken place rapidly, a red discoloration of the urine frequently follows.

In arriving at a correct diagnosis the student is greatly assisted by a process of elimination—that is, eliminating impossibilities and improbabilities, and so narrowing the issue down. For instance, in the early stage of the disease there is often great difficulty in determining whether a horse is suffering from biliary fever, a tick-transmitted disease, or horse-sickness, a disease

supposed to be transmitted by a mosquito or other night-flying insect. Now horse-sickness is a seasonal disease, and biliary fever occurs at all seasons of the year; therefore the season at which a doubtful case occurs may make it appear as almost an impossibility for it to be one of horse-sickness. Locality also plays an important part in this process of elimination, as there are places where horse-sickness rarely, if ever, occurs. Of course, this difficulty only exists in the early stages of the disease; usually within a few hours the typical symptoms of horse-sickness appear, making diagnosis an easy matter.

The history of the animal and the incubation period of the diseases also assist greatly in arriving at a correct diagnosis. To revert back to horse-sickness, the history of many cases that occur in towns and places where the disease is not usually met with reveals the fact that about ten days previously to the animal being noticed sick it went out with a picnic party into the country, or from other cause had a night in the veld. The history of the case may include a movement from one part of the country to another—for instance, if cattle are moved from tick-free veld such as the karroo to tick-infested veld (grass), and they sicken ten to fourteen days after arrival at their new home, the disease that is in all probability present is redwater. The period of incubation in such case rules out gall-sickness. In this connection, I may mention an interesting case that came to my notice with a small troop of cattle that were taken from a karroo farm and placed upon tick-infested veld. On release these animals were noticed to divide up into two mobs and graze separately, as it had evidently been their custom to do previously. In the one mob the majority developed visible symptoms of redwater, whilst the other mob remained, as far as could be observed, quite free of the disease. On enquiry it was found that the mob that contracted the disease had been bred and reared on the karroo tick-free portion of the farm, whilst the other mob were running on the irrigated grass veld tick-infested portion, and had evidently contracted the disease as calves, and the immunity then obtained had not broken down when subjected to reinfestation by ticks.

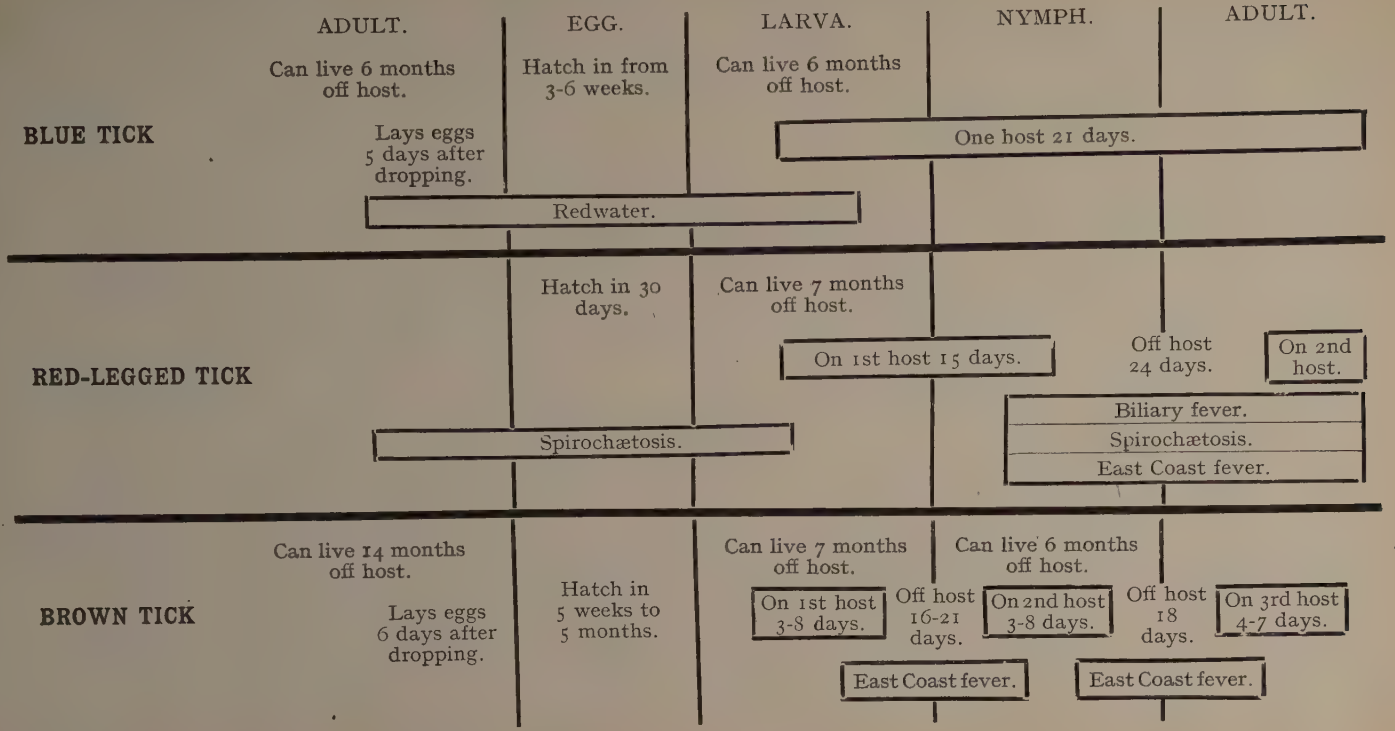
This process of elimination as an aid to diagnosis comes more or less as a matter of course to the practitioner, and is one that the student can profitably devote an amount of thought to; but the finding of the causal organism by microscopical examination is the final diagnosis in most of the diseases of this nature.

Reservoir of Disease.—A further similarity exists amongst these tick-transmitted diseases in that the recovered animal acts as a reservoir of the disease, and is capable of transmitting it by means of the tick to susceptible animals. Of course, immune animals get fresh supplies of the organism injected into them by means of the tick, but even without this source of supply it is probable that a limited number of organisms remain present and active in the system for a considerable time after recovery from an attack of the disease. In America it has been held that animals that have recovered from Texas fever or redwater, when placed upon tick-free country, harbour the organism in the system for a number of years. The exception to this rule of the recovered animal being a reservoir is held to be East Coast fever, and certainly, judging from the number of instances in which animals that have recovered from the disease and have run for lengthy periods in herds of susceptible and tick-infested cattle without an outbreak occurring, goes to prove that in a great number of, if not in all, cases that recovered, animals do not act as a reservoir of the disease. In a smaller number of instances recrudescences have occurred in such troops of cattle, and under such circumstances that it has been impossible to account satisfactorily for the outbreak except by the theory that a relapse of the disease has occurred in one of such recovered animals, by which it has been transmitted through the ticks to the susceptible animals in the troop or by the persistence of infectivity in animals suffering from a chronic form of the disease, such as occasionally is found in areas in which it has become endemic.

This persistence of the organism in the blood-stream or other portion of the animal's body leads to relapses in recovered animals when the system is subjected to severe strain or shock or other lowering influence such as occurs when the animal contracts another serious disease. In such cases the immunity to the existing disease may be broken down, and a relapse occur in which the symptoms of the old disease are present, as well as those of the more recently contracted one.

Perhaps this breaking down of the immunity has been observed more frequently in the case of redwater than in any other of the tick-conveyed diseases. It has been of common occurrence for animals affected with East Coast fever to exhibit all the typical symptoms of redwater, including the passage of bloody urine, and for the organism to be demonstrated in numbers in the blood. Rinderpest and the preventive inoculation for same

DIAGRAM SHOWING THE STAGES IN THE LIFE CYCLE OF THE BLUE, BROWN, AND RED-LEGGED TICKS.



also frequently break down an animal's immunity to red-water.

It is possible for other causes than disease to cause a relapse, and in this connection may be noted the deleterious effect that wetting of the body has upon the subject of not only tick-conveyed diseases, but also upon other tropical diseases of a similar nature. It has been recognised for a great number of years that a person harbouring the malarial fever parasite had to avoid getting wet, or a relapse of fever would occur, also that animals bitten by the tsetse fly and suffering from nagana or trypanosomiasis would linger on through the dry season until the first rains, when a number would succumb. The same occurred with transport animals suffering from this disease if they got wet when crossing a river, and it has been observed that the dipping of animals suffering from tick-conveyed diseases quickly develops the typical symptoms of the disease.

EAST COAST FEVER

This disease was known in South Africa as Rhodesian redwater until 1903, when Professor R. Koch came out to Rhodesia to investigate it, and having discovered the true nature of the disease, he named it "African Coast fever," which name is frequently used to designate it at the present time; but latterly the name "East Coast fever" has been more frequently used, and inasmuch as the disease is one belonging to certain parts of the eastern coastal regions of mid-Africa, the latter is perhaps the more appropriate name for it.

History.—The disease was introduced into Rhodesia and what is now the Union of South Africa by a shipment of cattle from Dar-es-Salaam in German East Africa. This shipment left port in the latter part of 1901. It is alleged that a number died on the passage down; part of the consignment were landed at Beira, and the balance went forward in the ship to Delagoa Bay. The history and the progress of the disease in what is now the Union of South Africa was similar to what it was in Rhodesia, but as I am better acquainted with its developments in the latter country, I shall deal with them there only. Of the animals landed at Beira, several truckloads were sent forward by train to Umtali, and of these some went on to Salisbury. The disease broke out in both towns, but its nature was not recognised, principally on account of the fact that in a great many instances, at the onset of the disease, when an animal immune to redwater (which all Rhodesian cattle were in those days) contracts East Coast fever, the immunity is broken down, and the symptoms shown were in many cases principally those seen in redwater, and it was thought to be a severe form of this disease. Dr. Koch originally held a similar view, for when he investigated the disease in German East Africa in 1897, in his report, although he describes the causal organism, he apparently at that time did not regard the disease as separate and distinct from redwater, but spoke of mild and severe forms of the latter. However, the thought soon gained ground, and was borne out by the post-mortem appearances, that the disease was something more than

redwater, even though in many instances it was complicated with it. The need for further investigations became apparent, and in 1903 the eminent bacteriologist, Professor Koch, was obtained by the Rhodesian Administration, and he came out and studied the disease in Rhodesia.

Although Dr. Koch showed that East Coast fever was not a virulent type of redwater, his conclusions in many respects were incorrect, as he maintained that the blue tick was responsible for the spread of the disease, and that the disease passed over from the infected adult tick to its progeny; while it was subsequently shown that the method of inoculation recommended by him as a protective measure was entirely ineffective.

For some months after the disease broke out at Umtali and Salisbury it was confined to stock which had grazed on the infected veld at those centres, but soon it started spreading by means of the oxen used for transport along the main roads throughout the country. It radiated out from Umtali to Melsetter, was present along the main road from Umtali to Salisbury, thence to Gwelo, and finally reached Bulawayo, and radiated out again from these towns to mining and other centres that drew their supplies by ox-waggon from the towns. From Bulawayo it spread to Gwanda and Belingwe, but by the end of 1902 more knowledge of the disease had been obtained. An attempt was made to stop it at the Shangani River, but was too late. The movements of cattle in infected areas in Matabeleland were restricted, and permits had to be obtained to make them. Dipping tanks had been established at Bulawayo and Salisbury, and transport cattle were compelled to be dipped, if they came upon those respective commonages, at least once every three weeks. The mines had to be kept going; therefore the roads leading to them were declared infected areas for one mile on either side. Areas around the towns and the mines were also declared infected, out of which no cattle were allowed to move; consequently it came about that the disease did not spread south of Bulawayo towards Plumtree or north towards Victoria Falls, and it was almost without exception confined to the declared infected areas, including the two miles wide roads. In the course of a year or two most of the cattle on these areas were dead; only about 5 per cent. remained in infected herds, and when the ox transport was replaced by mule and donkey transport and railways, a noticeable lull in the disease occurred. This was followed by recrudescences of the disease amongst previously infected herds, from which the disease in many cases again radiated. Such

outbreaks have continued at different times right up to the present day, but during the last two years a few outbreaks have occurred at places difficult or unable to be connected up with previous infection.

Nature.—The disease is not directly contagious; a sick animal will not infect a healthy one by contact, neither will any of its excretions or fluids from its body. In nature an intermediary host is required for the transmission of the disease from one animal to another. This host is found amongst two or three species of ticks, but the brown tick is the principal transmitter of the disease, which is effected this way: If a tick in its larval stage feeds and engorges upon an animal sick with the disease at the time that the parasite causing the disease is present in the blood, it can transmit the disease to a healthy animal in its next stage (nymphal); or if it feeds on a sick animal in the nymphal stage of its existence, it can transmit the disease in its adult stage. The disease does not pass from the adult tick through the egg to the larvæ, as is the case with redwater in the blue tick; and from experiments carried out by Sir Arnold Theiler, it was found that once a tick had bitten it discharged its poison, and was no longer capable of infecting, also that one tick might in a certain number of cases fatally infect an animal, but two were quite sufficient to do so in every instance. The brown tick is an intermittent feeder—that is, it drops off its host when engorged at the end of each stage of its life, changes its skin, and then seeks another host for its next stage. This changing of the skin, or moulting, takes usually about three weeks, but it may take up to eight. Practical demonstrations of this were given with outbreaks of the disease in 1902, when the first case was detected; this was followed in six to eight weeks with quite a number of animals affected.

The disease is caused by an intracorpuscular parasite called the *Piroplasma parvum*, which does not destroy the corpuscles of the blood.

Symptoms.—The first indication of the disease is a sudden rise in temperature, which may reach as high as 107° F., even while the animal may remain apparently healthy; but as owners do not take the temperatures of their cattle at ordinary times, this symptom is useless until the disease has been discovered in the herd and temperatures are regularly taken, as, in spite of this rise of temperature, the animal will continue feeding and chewing the cud and show few actual symptoms until the disease is far advanced.

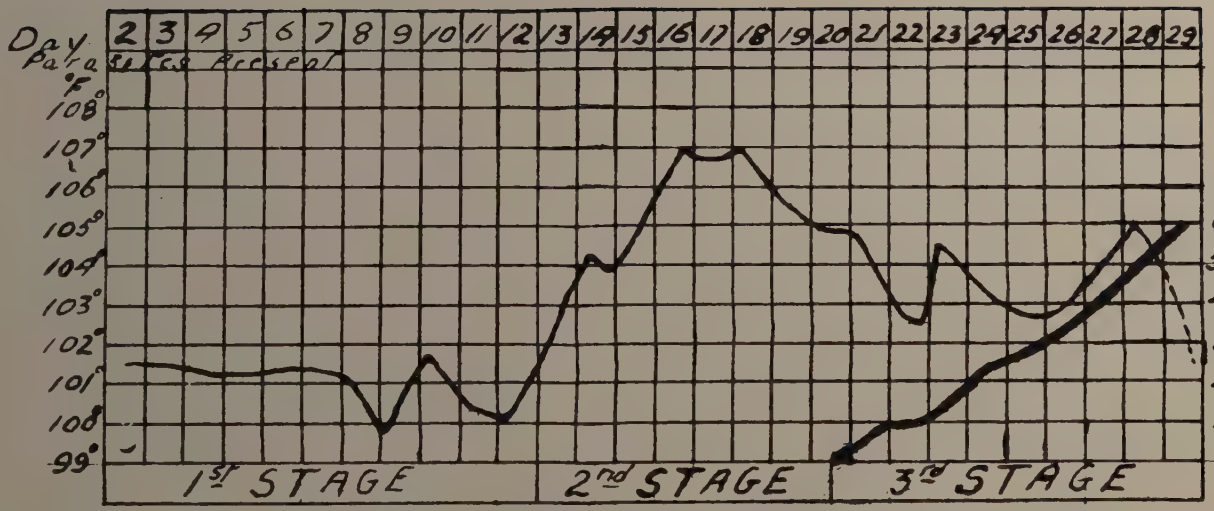


FIG. 10.—CHARACTERISTIC CHART OF AFRICAN COAST FEVER.

The chart shows the body temperature of a cow infected in England by ticks sent from South Africa. The thin black line indicates the temperature record. The rise in the percentage of infected red blood-cells is indicated by the heavy black line.

Stage 1.—The period of incubation.

Stage 2.—The period when the temperature is elevated, but the parasites are not present in the peripheral blood. Koch's granules may be found in the spleen and glands.

Stage 3.—The period when the parasites are present in the peripheral blood.

Amongst the earlier symptoms that can be noticed in an animal are specks of blood and streaks of mucus on the dung; this is frequently normal in consistency, but the animal may be constipated at first, followed by diarrhœa. If an animal in this early stage of the disease is watched as it feeds along with the rest of the herd and slowly followed up, sooner or later, in the majority of cases, it will be seen to separate out from the herd and stand under a tree or bush, when the first real symptoms of the disease will be shown: the ears will drop, and there will be an occasional dribble of saliva from the mouth; but it should be

FIG. 11.—EAST COAST FEVER (ANIMAL DEAD), SHOWING FROTH FROM NOSTRILS.

(Post-mortem showed extensive lung lesions.)

approached quietly, for if the animal is disturbed it will walk off and feed again with the others as if nothing was the matter.

The symptoms from then on are progressive: the animal looks dull, and a falling off in condition is noticeable; a running from the eyes, nose, and mouth is seen without difficulty; a husky cough may be present, the eyes appear sunken in the head, there is a swelling of the glands of the throat and neck, also those in the flank, coupled with a weakness in the loins and a staggering gait.

Post-mortem Appearance.—When the disease is complicated with redwater, as it frequently is, many appearances of the latter disease may be present. The most important lesions to look for in East Coast fever are situated in the glands, the fourth stomach, and the kidneys.

The fat of the carcase is usually of a very yellow colour, and between the muscles a yellow-coloured fluid is found; this fluid is frequently found in the cavity of the chest and in the lungs, giving them a dark-coloured and a sodden appearance. If this fluid gets into the bronchial tubes and windpipe it will escape from the nostrils at the time of death in a froth, similar to what occurs in horse-sickness

The lymphatic glands, commonly called kernels, will be found to be enlarged, and those connected with the lungs and intestines, instead of being pale yellow or whitish-yellow, will be congested and purple in colour. The lining of the fourth stomach is inflamed and frequently ulcerated. After the thin skin, called the capsule, covering the kidney has been removed, small yellow or white spots called infarcts will be seen, which stand up from the surface of the organ; these, when cut into, are found to extend into the kidney, ending in a point, making them roughly triangular in shape and about the size of a pea or smaller in dimensions. In their earlier stage these infarcts are often hæmorrhagic. Blood-spots may also be found on the heart and on the intestines; the gall is usually very thick, but if infarcts are found in the kidneys they may almost be regarded as diagnostic of the disease. Occasionally the bowels are ulcerated, and sometimes contain long clots of blood.

Diagnosis.—The final diagnosis rests with the finding of the organism under the microscope; therefore in the living animal take blood-smears from the ear, and in the dead animal spleen pulp smears as well as blood for examination.

Treatment.—The treatment of the disease has been based upon the following facts:

After an animal has been bitten by infected ticks, about ten or twelve days elapse before a rise in temperature, which is the first symptom of the disease, takes place. Simultaneously with this rise a form of organism known as "Koch's bodies" are to be found in the lymphatic glands, and this condition of high temperature and Koch's bodies in the glands continues from three to eight days before the parasites appear in the blood, which they do usually about the twentieth day; therefore it follows if the disease can be detected by the elevation in temperature,

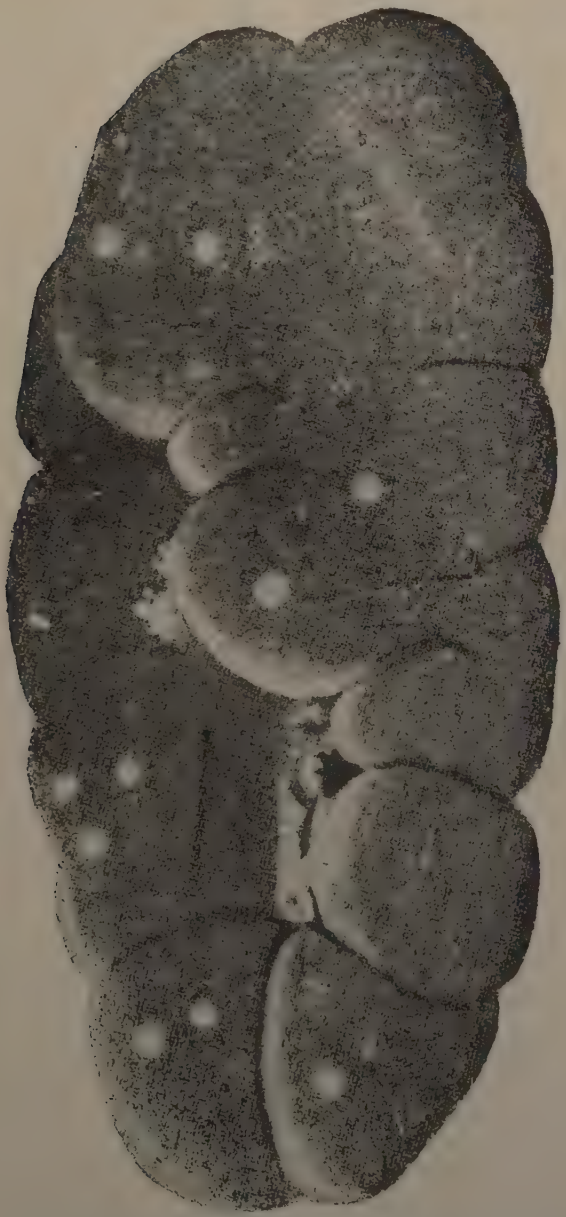


FIG. 12.—KIDNEY FROM CASE OF AFRICAN COAST FEVER, SHOWING THE AREAS OF NECROSIS (INFARCTS) IN VARIOUS STAGES. (W. Robertson, M.R.C.V.S.)

the glands be punctured, a smear from same be examined under the microscope, the presence of Koch's bodies demonstrated, and the animal be killed before the parasites get into the blood-stream and so infect the ticks, one step in the eradication of the disease will have been effected.

When attempts were first made to eradicate the disease from infected herds this knowledge was acted upon; the herd was temperatured, the sick animals were destroyed, the remainder were then removed to fresh grazing and kraals, ticks were kept down by weekly sprayings with a 25 per cent. solution of paraffin and water or an arsenical dipping fluid, the temperature was taken night and morning, and animals showing a persistent rise in temperature were destroyed. The cattle remained in their new quarters for twenty-one days. If cases occurred during that time the animals were again sprayed and moved; this process was repeated until the disease had been left behind, which it usually was with the second removal. This system was found to be efficacious, but it had many drawbacks, amongst which may be mentioned one—the uncertainty when moving the cattle that the veld they were taken to was not infected; then there was the land left behind with infected ticks on it, into which beasts might stray in spite of attempts to keep them off it. This land was quarantined for about eighteen months, during which time the ticks would have become attached to some other animal, and so discharged their poison or have died from starvation. Perhaps the worst feature of this method was the area of land required to move cattle over; it could only be practised in thinly stocked areas.

Fencing had early been recognised as valuable in preventing the spread of, and as an aid in eradicating, the disease; likewise keeping the animals in the stables had been adopted to save cows for milk-supplies, also calves.

In 1910 Watkins Pitchford, who had been working in Natal with a view to eradicate the disease by destroying the ticks, published his report and introduced the best method of dealing with the disease that has yet been found. In studying the life history of the brown tick, he discovered that in exceptional cases a tick might only remain on a beast as short a time as three days. His next step was to find a dipping fluid that would kill ticks, and in which it was safe to dip cattle every seventy-two hours; this he was successful in doing, and recommended a dipping fluid composed of 4 pounds arsenite of soda (80 per cent. arsenic), 3 pounds soft-soap, 1 gallon paraffin, 400 gallons of water.

His method is the one now successfully practised to eradicate

the disease from a herd. The cattle are dipped every seventy-two hours; the ears and tails are dressed to destroy the ticks attached to these places; temperatures are taken and sick animals destroyed. The cattle are moved from infected grazing on the farm where possible, and other non-susceptible stock sent there to pick up the ticks; if not, the animals remain on the infected grazing. Whether the method will prove the final one for eradicating the disease from the country is doubtful, but remains to be seen.

PIROPLASMOSIS OF CATTLE

Synonyms.—Redwater (South Africa), Texas Fever (America), Tick Fever (Australia), Tristeza (Argentina).

History.—The disease is world-wide in the sense that it occurs in all the continents. It was first observed in South Africa in 1870, in Natal, at the Tugela River, which forms the boundary between Zululand and Natal. How it got there or whence it came has never been clearly elucidated. One belief was that it came from Swaziland; another was that it was due to the importation of cattle from Madagascar, or from the east coast. It may have been introduced by either of the ways suspected.

The disease travelled along the coast districts of Natal to Durban, and, following the main lines of ox-waggon traffic, it had penetrated as far as Kokstadt, in Griqualand East, in 1873. The Pondos to the east kept it back for a number of years, as they permitted no oxen to go through their country, but supplied healthy cattle for hire to the traders, until in October, 1882, the Cape Government sent up a newly formed regiment, the Cape Infantry, and forty waggons of baggage, with authority to go on to Kokstadt, the transport riders holding a guarantee that all cattle which died of redwater would be paid for out of the public purse. A number of oxen died on the return journey, and several in the Kaffrarian districts. By this means the disease spread rapidly in the Transkei country, extending along the coast in a southerly direction.

While the disease was spreading in the south, it was extending quite as rapidly towards the north-west. In the space of three or four years it had entered the Free State and the Transvaal. The opening up of the interior of the country by the diamond and gold mines disseminated the disease still further, and the Boer War in 1900 scattered it far and wide, until at the present time it is practically present in all portions of the sub-continent where ticks prevail.

How the disease reached Rhodesia has never been definitely decided. Some hold that it was introduced by transport oxen from the south, many of which came from Natal at the time of

the occupation of the territory by the British; but so far as can be judged, the disease was enzootic in the country previous to that time, and had probably been in existence for a number of years, the same as it had been in the adjoining countries bordering the east coast.

The disease was first studied bacteriologically in Roumania by Babès (1888); shortly after him (1889) Smith and Kilborne in North America advanced the knowledge by the discovery of the fact that the nature of the organism causing the disease was a hæmosporidium, and that it was transmitted by a tick. Following these investigators came a number of others in various parts of the world. The next most notable fact in connection with the disease was that of Sir Arnold Theiler in 1908 by the discovery that redwater or Texas fever, as hitherto described, covered two diseases that may exist separate and distinct from each other, but that are usually present at the same time in the animal; these are redwater, which is caused by the *Piroplasma bigeminum*, and gall-sickness, caused by the *Anaplasma marginale*. This view did not find favour, and was not accepted by all scientists at first. Many preferred to retain the old belief that *A. marginale* was only an altered form of *P. bigeminum;* but upon further experimental proofs being produced, the theory is now generally accepted.

Nature.—The disease cannot be regarded as a contagious one in the ordinary sense in which the word is used, as in nature the presence of the tick is necessary to transmit the disease from the affected to the susceptible animal. The disease occurs usually as an enzootic, when it affects calves during the first few weeks or months of their existence, and usually runs a very mild course. In many instances the symptoms are not noticeable in animals of the native and Africander breeds of cattle, but with the progeny of animals imported from Europe the symptoms are more pronounced. This holds also to a greater or less extent with the progeny of a well-bred bull out of native cows, and apparently the virulence of the disease is also intensified by transmission through susceptible animals. It may also be regarded as somewhat seasonal in character, although it can be contracted at any season of the year. The cases are fewer and milder in character in the winter season than those that occur in the summer. The number of ticks also determines to some extent the virulence of the attack; the greater the number of ticks present on an animal, the more severe the resulting disease.

Age also plays an important part as a factor in governing the severity of the attack, young animals having greater resistance,

or perhaps it would be more correct to say recuperative powers, than mature or aged animals. The question of age is one for consideration when importing pure-bred animals from non-redwater countries; the younger the animal, the better chance it has of recovering from the disease.

An animal that has recovered from redwater is immune to the disease; this immunity is intensified where animals are kept on tick-infested pastures by the transference of the parasite by means of the tick from one immune animal to another, also from calves suffering from the disease to older immune cattle. The degree of immunity naturally acquired is influenced by the fact of the existence of different strains of the disease in different localities, and even on different farms in the same locality; therefore it comes about that animals immune to redwater in one locality, when removed to another, may have their immunity broken down and suffer from a relapse of the disease.

Even without reinfection by ticks, the blood of animals that have recovered from redwater remains infective for a considerable time; and while, so far as I am aware, no experiments have been carried out in South Africa to prove the extreme length of time that the blood may remain infective, it has been proved that this may extend to a period of at least two years.

Symptoms.—The first noticeable symptom will be that the animal appears to be dull and sluggish, and has a disinclination to move and stands apart from the rest of the herd; the hair stands erect; the ears droop and the eyes are dull. On taking the temperature, it will be found to be high—104° to 107° F. The animal ceases to feed or ruminate, and falls off rapidly in condition; later on, if compelled to move, it walks with a straddling gait, as if weak across the loins; the muscles behind the shoulder and in the flank are noticed to quiver, the pulse and breathing are quickened, and as the disease progresses the latter becomes laboured; finally, the animal lies or falls down, becoming semi-conscious, and with grunting respiration. The bowels at the onset of the disease are often normal; at times there is a tendency to diarrhœa, but as the disease progresses the fæces become harder and of a brownish colour. The diagnostic symptom is the passing of bloody-coloured urine; this is not present in all cases, even in some of those that terminate fatally; but it is usual, according to the severity of the attack, to have the urine discoloured by the broken-up blood-cells, and varying in colour from a light claret to the colour of coffee. In animals recovering from the disease symptoms of anæmia and jaundice are usually

well marked. Pregnant animals frequently abort, and these cases are usually fatal.

Post-mortem Appearances.—The spleen is enlarged usually three or four times its normal size, and its contents are pulpy, resembling blackberry jam. The third stomach (leaf stomach) is usually dry and hard, and the fourth or true stomach is congested and of a purplish-pink colour; this congestion extends to a greater or less extent through the bowels, which are bile-stained. The liver is enlarged and saturated, oozes when cut, and is friable; the gall-bladder is mostly distended with bile; the urinary bladder should always be examined for dark-red-coloured urine. The lungs are generally healthy. The heart usually has ecchymoses (blood-spots) upon its inner or outer surface. The kidneys are congested and dark in colour.

Incubation.—The incubation period of the disease is from five to fourteen days.

Treatment.—A successful treatment has been found in the subcutaneous or intravenous injection of trypan blue. The effect of this drug is to destroy the organism in the blood, but it has been found that it does not do this so completely as to destroy the immunity of the animal to the disease if it recovers; still, its effect is most marked. The animal may be in a very serious condition, with a temperature of 107° F. when injected, and the next morning the temperature will be found to have fallen to quite or nearly normal, the appetite returned, and every indication of a quick recovery to health. The effect is more rapid when the drug is injected intravenously, but in ordinary cases, and where death is not apparently imminent, the subcutaneous injection is usually adopted. The site selected is either behind the shoulder or in the dewlap, but as there is a great tendency to abscess formation after such injection, although antiseptic precautions may have been carried out, the dewlap is the better site, as it allows of a freer discharge of the abscess contents.

It is important to remember that only freshly prepared solutions of trypan blue should be used; a 2 per cent. solution is the strength usually employed, and the drug can be obtained in tabloid form, with directions for making a solution of this strength. By adding 7 ounces of boiling water to 1 dram of powdered trypan blue a 2 per cent. solution is prepared, which, upon cooling, may be injected in the following quantities:

Calves up to nine months old	50 c.c.
Young stock nine to eighteen months old	100 c.c.
Full-grown cattle	150 to 200 c.c.

One injection only is required, and no good will be obtained by repeating it. If the animal is constipated, a dose of Epsom salts, linseed oil, or calomel will assist, but the dose should not be an excessive one. If the sick animal is anæmic and has but a poor appetite in the convalescent stage, nux vomica will help it. This may be given as 1 dram of powdered nux vomica in gruel or 1 ounce of the tincture twice a day; mild stimulants such as ale and stout also help good nursing to effect a speedy recovery. Defibrinated blood taken from a healthy animal is sometimes administered as a tonic, with good results.

PIROPLASMOSIS OF HORSES

Synonyms.—Biliary Fever (Dutch, *Wit-beek ziekte;* French *Piroplasmose equine;* Italian, *Febbre tifoide*).

History.—The disease occurs all over Africa; it has also been studied in Italy, Russia, and India. Dr. Koch studied this disease in East Africa, also to a small extent when he was in Rhodesia in 1902 investigating East Coast fever. At that time a great number of donkeys were imported into Rhodesia from the southern States; many of these animals came from the karroo, where the disease was not in evidence, with the result that the disease was very prevalent amongst these animals, and was attended with fatal results in a number of cases. The disease was called *wit-beek ziekte* (white-mouth sickness), for the reason that this is one of the most pronounced symptoms of the disease in donkeys. But it was the investigations of Sir Arnold Theiler later on that furnished a greater amount of knowledge of the malady.

Nature.—In nature the disease is essentially similar to the rest of the diseases due to piroplasms, in that it requires a tick (in this case the *Rhipicephalus evertsi*) for its transmission from one animal to another, and in being caused by an organism that lives inside the red corpuscles of the blood, which eventually causes the death of the corpuscles, and by their destruction and the generation of a poison (toxin) produces the symptoms peculiar to this disease.

The disease affects horses, mules, and donkeys, and the organism has been found in the blood of the zebra. Animals born in places where the disease is enzootic contract it early in life, recover, and become immune, many passing through it without showing any visible symptoms. It would appear that breeding also increases susceptibility to a certain extent, not so much so, perhaps, as in redwater, neither does age affect the issue to the same extent; a greater percentage of aged animals recover from an attack of biliary fever than is the case in redwater.

It would appear, also, as if different strains of the disease are

in existence, as animals taken from one tick-infested portion to another may suffer from an attack of the disease, usually a mild one. Relapses of the disease also occur.

Symptoms.—The first symptom noticed as a rule is that the animal is dull and sleepy; this will be observed first if the animal is in regular work. In the stable it will stand with the head depressed, the ears dropping, the eyes half-closed, presenting a very sleepy appearance. The temperature will be found to be high—103° to 106° F.—with the pulse accelerated and the mucous membrane of the eye and mouth tinged with a yellowish colour, or congested and tinged with yellow. The mouth has an offensive odour. The urine is usually increased in quantity and of a high colour; in a small percentage of cases it may be actually red, similar to redwater in cattle, but it is more frequently bile-stained. The bowels are usually somewhat constipated, the fæces being dark yellowish-brown and coated with mucus. In the mucous membrane of the eye blood-spots are often seen; these may run together, forming large irregular blotches. There is frequently swelling of the eyelids. The animal loses condition rapidly, and at the end of a case that even terminates very favourably emaciation is usually marked. Many cases run a mild course, and recovery follows in a few days. Amongst the unfavourable symptoms are the temperature remaining high—105° to 107° F.—for several days, persistent entire loss of appetite, the mucous membranes remaining a dirty brick-red colour and not clearing up (the usual way is for the membrane, after the onset of the disease, to assume a pale anæmic colour, which gradually becomes tinged with pink as health returns), very rapid loss of flesh and emaciation, the animal becoming very weak, staggering and tottering when it moves, perhaps actually falling down in a semicomatose condition. Animals that develop such symptoms may recover, but frequently the process is a protracted one, the animal remaining weak and debilitated for a considerable time. In other cases the animal lingers on, death ultimately resulting from general debility.

Post-mortem Appearances.—A jaundiced condition of all the organs of the body is the most noticeable post-mortem feature.

The liver is enlarged and of varying colour (yellow or brown) and consistence, depending on the severity and duration of the disease; the spleen is double, or even more than that of its normal size; the heart is pale and softened, and can be pulled to pieces easily with the fingers in some cases; the kidneys are also pale and friable; the lymphatic glands are swollen and œdematous;

blood-spots are frequently found upon the heart, and a quantity of straw-coloured fluid may be present in the heart sac. The lungs are sometimes œdematous and infiltrated with serum to such an extent that death is in such cases at times attributed to horse-sickness.

Incubation.—By inoculation an incubation period of seven to twelve days elapses before a rise in temperature and the first clinical symptoms manifest themselves. In naturally acquired cases of the disease a similar or perhaps slightly longer interval occurs.

Treatment.—Some veterinarians have reported excellent results from trypan blue treatment in certain cases, while others are equally emphatic in asserting this drug is entirely ineffective. Investigations carried out at the Veterinary Research Laboratory, Pretoria, appear to indicate that there are two types of equine piroplasmosis in South Africa, and that while trypan blue is effective in the treatment of one type, it is ineffective in the other. For this reason it is well in all cases to inject a dose of trypan blue, giving 100 to 150 c.c. of the solution prepared as prescribed in the section on Piroplasmosis in Cattle.

A recent treatment that has been found beneficial is the daily injection subcutaneously of 10 c.c. of a 2 per cent. solution of hydrarg. perchlor.

Particular attention should be paid to good nursing and the food, even if the animal retains an appetite; all hard foods—oats, maize, forage, etc.—must be discontinued, and a diet consisting of bran, greenstuff, and suchlike soft foods be substituted. The bowels should be watched carefully, and to counteract the tendency to constipation a linseed mash is useful. Boil a pint of linseed (the seeds) in a gallon of water, let it simmer slowly for several hours; when it is cooked, mix half of it with bran and give as a mash. The balance can be given the following day, or a bottle of raw linseed oil may be given in place of the mash; and if the constipation is at all severe, ½ dram of calomel may be given as well as the oil.

Following treatment to get the action of the bowels corrected, methylarsenate of soda can be employed, given in doses of from 15 to 30 grains, according to the size of the animal, once daily for a few days; but the administration of this drug should not be pursued for a lengthy period, and if purging supervenes, indicating that arsenical poisoning is approaching, it should be discontinued at once, and flour gruel be given in a bucket to drink to counteract the diarrhœa. In the convalescent stage, if the appetite is

not very good, a tonic may be given. The following is a good one: ½ ounce each of tincture of nux vomica, tincture of gentian, and liquor arsenicalis, given in a bottle of oatmeal night and morning. Stout is also a good stimulant and tonic, but if the animal is very anæmic there is nothing better than defibrinated blood, prepared and administered as described in the treatment of gall-sickness. I have seen horses eagerly take this, and lick odd drops that have been spilt.

PIROPLASMOSIS OF DOGS

Synonyms.—Biliary Fever in South Africa (French, *Fievre bilieuse, Jaunisse maligne*).

History.—The disease was first discovered in Italy, then in South Africa, where a considerable amount of investigation has been devoted to it.

Nature.—It is similar in all respects to the other diseases belonging to this family. It is principally a disease of tropical climates, and is particularly prevalent in India and China. It is probably transmitted by different ticks in different parts of the world; the tick that is responsible for its transmission in South Africa is the *Hæmophysalis leachi*, or dog tick. Dogs are apparently liable to a second attack of the disease if they are transferred from one part of the country to another, and that, lowering the vitality of the animal, may result in a relapse of biliary fever. In clinical observation it would appear as if "dog distemper" is capable in some cases in bringing this about, and in bad years for the disease cases are met with in old dogs which it is highly improbable have escaped previous infection.

Symptoms.—The most noticeable symptoms are the extreme languor and the entire loss of appetite, the temperature elevated, the nose hot and dry, the mucous membranes of the mouth and eye congested and tinged with yellow, and later becoming anæmic and decidedly yellow in colour, the breathing accelerated, the urine high-coloured, frequently red, and at times a greenish coffee colour.

Incubation.—The incubation in naturally acquired cases is from seven to fourteen days.

Treatment.—Until Nuttall and Hadwen discovered the treatment of trypan blue, this disease was a veritable scourge to the dog-owning community of South Africa, and a nightmare to the veterinary practitioner; but at the present time, if a case is treated by this drug before the animal is *in extremis*, a favourable result may be anticipated. From 5 to 15 c.c. of a 2 per cent. solution injected either intravenously or subcutaneously is a sufficient dose for dogs ranging in size from a large fox-terrier

up to a St. Bernard. It is advisable to err on the side of a large rather than a small dose; for small or toy dogs a 1 per cent. solution is recommended (to prepare a 2 per cent. solution see Redwater). To obtain a quicker result the injection may be made intravenously, the vena saphena being a convenient site; but quite good results are obtained by subcutaneous injection, the inside of the thigh being a favourite spot. This should be first thoroughly cleansed; the ordinary tincture of iodine rubbed on with cotton-wool answers admirably. The only objection to the subcutaneous injection is that an abscess is liable to form, but in the site mentioned it is of little importance, and only requires opening to allow the contents to escape. About twenty-four to forty-eight hours after this treatment a great improvement usually takes place in the patient, the appetite returns, a wagging of the tail denotes the animal is beginning to take interest in life again, which may not on the whole be so bad as to be not worth striving for, and usually the road to recovery is unhindered.

ANAPLASMOSIS IN CATTLE

Synonyms.—Gall-sickness and Bush-sickness (South Africa).

History.—Sir Arnold Theiler was the discoverer of this disease. In 1910 he was enabled to separate it from piroplasmosis or redwater. Until that time the disease and the symptoms it gave rise to had been regarded as a secondary reaction due to *Piroplasma bigeminum*, and the causal organism *Anaplasma marginale* was considered an altered form of that organism; but by experimental inoculation Theiler was able to produce in an animal either redwater or gall-sickness independent one of the other. The previous confusion had been caused by the blue tick in the majority of cases transmitting the two diseases simultaneously into the animal, and the difference in the length of the incubation of the two diseases not being correctly understood.

Like redwater, the disease is found all over Africa, and the causal organism has been described as existing in North America, South America, and the Transcaucasus. It is also believed to exist in Italy, but it has not yet been demonstrated as existing in India, China, or Queensland, where the piroplasma exists.

Nature.—Like redwater and most other tick-conveyed diseases, this disease is one in which the red blood-corpuscles are destroyed to a great extent by the organism, and also one in which an intermediary host is required to transmit the disease from one animal to another. Like redwater, too, the disease is usually contracted by calves at an early age, and causes as a rule little disturbance to native and Africander cattle; but the disturbance is greater the higher the animals are graded up by European breeds of cattle. The disease can occur at any age in life, the rule being that the later it occurs the more severe are its effects. Clinical experience goes to prove Sir Arnold Theiler's findings, in that one meets with numbers of cases of gall-sickness in which no trace of redwater can be found, and at certain seasons, for some unknown reason, the disease is much worse than in others.

Symptoms.—The first clinical symptom is a fever reaction, but this may be present for some days before the animal is

noticed to be sick. Even with a temperature as high as 107° F., the first thing to be actually noticed may be that the animal is capricious in its appetite or off its food, or that it is constipated and passing hard, yellowish-brown fæces in lumps somewhat resembling those of a donkey's in size and shape. The eye usually has a wild, staring look, and many animals become aggressive and inclined to charge the attendant. A most noticeable symptom to the careful observer is the rapid loss of flesh; this may be so pronounced as to be seen from day to day. The symptoms of anæmia are also very pronounced in the bloodless, white colour of the eye and mouth, accompanied by jaundice, denoted by a yellow coloration, not only in the sites mentioned, but throughout the body, and can be easily seen in those parts, such as the ears, udder, and thighs, where the hair is not thick; and in bad cases in white-haired animals the yellow-coloured skin will show through the hair, and the scurf from the skin will also be yellowish in colour. The muzzle is dry, at times cracking, and there will frequently be a dribbling of saliva from the mouth. The urine is distinctly high-coloured, but never blood-stained; quivering is often found present in the muscles of the shoulder or the flank. A peculiar grunting sound accompanies each respiration, and when the disease has far advanced towards a fatal issue the animal is usually found lying down, making this peculiar grunt, and with the eyes sunk deeply into the head; but animals showing all these unfavourable symptoms may recover from the disease.

In conclusion, it is to be noted that there is little difference in the foregoing symptoms to those described in redwater, and, as stated previously, to arrive at a correct clinical diagnosis the process of elimination has to be considerably employed. One certain difference is that blood-tinted urine is never found in gall-sickness, and usually the nervous symptoms, the staring eye, aggressiveness, and trembling, are more pronounced in gall-sickness than in redwater.

Post-mortem Appearances.—The condition of the carcase will depend upon the length of time the animal has been ill; if for only a short time, the wasting will not be so pronounced as it is when the animal has been ill for some days. The blood will be seen to be thin and watery, and oozing out of the small blood-vessels as the carcase is being flayed. The flesh will be a pale, bloodless colour, the fat distinctly yellow, giving the carcase a yellow colour, and usually infiltrations of a jelly-like substance are found between the muscular layers, particularly in the lower

parts of the abdomen and brisket. The liver is enlarged frequently to a considerable extent, and appears swollen and distended, and on cutting into it it will usually be found to be of a deep brown or saffron yellow colour.

The gall-bladder is commonly distended with thick bile. The spleen is considerably enlarged, the pulp being thick and soft, resembling jam. The third or leaf stomach is usually dry and hard, the fourth stomach slightly congested, the bowels showing similar patchy congestion, and containing bile-stained food; the bladder contains normal or high-coloured urine. The lungs are frequently œdematous, and frothy fluid exudes from them. When they are cut with a knife, a variable amount of straw-coloured fluid is usually found (if the animal has been ailing for some days) in the cavity of the chest and in the heart sac. The heart is often very pale in colour, with blood-spots (ecchymoses) upon it.

Incubation.—By inoculation with immune blood there is a period of incubation of from twenty-seven to thirty-two days; with virulent blood about sixteen days. By tick infestation it is longer—about forty-two to fifty days.

Treatment.—No drug has yet been discovered that is capable of destroying the *Anaplasma marginale* similar to the way trypan blue destroys the piroplasma in cattle, and when this drug has been tried for gall-sickness it is open to doubt if its effects are not more deleterious than curative. Consequently a great number of treatments are adopted, but the remedial effect of most of them is very doubtful. The treatment that apparently meets with the most success is, in the first place, to get the bowels opened with a purgative of Epsom salts, calomel, or linseed oil, either one or the other or all being used before the desired result is obtained. The bowels are in a state of torpor, and considerable quantities of purgatives may be given without result. In such cases the administration of a stimulant such as whisky, gin, or brandy may assist. If the bowels are opened a favourable result is anticipated. This treatment may be followed by the administration of methylarsenate of soda, once a day for three days (only), in doses of from 10 to 60 grains according to the size of the animal, ranging from a two or three months old calf up to a full-grown beast. Others claim good results from giving 20 to 25 drops of croton oil as soon as the animal is noticed sick. The old remedy of the transport rider, which is still frequently used, is a tablespoonful of muriatic acid (acid. hydrochlor. dil.) in a bottle of cold water.

The only treatment that can be spoken about with assurance is, when the animal has recovered from the acute stages of the disease and is in a weak, debilitated condition, the administration of two or three bottles of defibrinated blood daily, which helps greatly in restoring the strength and appetite.

The blood of any animal will apparently do. All that has to be done is to " whip " it when freshly drawn with a wisp of wires or twigs to remove the fibrin, and so prevent it clotting.

ANAPLASMOSIS IN SHEEP

Gall-sickness is a term used by the South African farmer to include other diseases than the disease that is caused by *Anaplasma marginale;* in fact, the term may be taken to include

FIG. 13.—ANAPLASMOSIS OF SHEEP (NATURALLY INFECTED LAMB), SHOWING THE CONDITION KNOWN AS " BOTTLE-JAW."

any disease in which the post-mortem examination reveals a distended gall-bladder and a jaundiced condition of the carcase, if there is no diagnostic symptom, such as red urine, of any other known disease present. Therefore it does not appear strange that a disease or diseases should be known as gall-sickness in

sheep, but there is a disease affecting sheep that is so similar to anaplasmosis in cattle, both in its ante-mortem symptoms and its post-mortem appearances, that it has come to be regarded as one of the plasmoses. Bevan (Rhodesia) corroborates this clinical observation in his report for the year 1912. He states:

"*Anaplasmosis of Sheep.*—Early in the year it was discovered that in many districts sheep thought to be suffering from verminous disease were affected with anaplasmosis. Some experiments were conducted to study the course of the disease in sheep."

The results of the experiments mentioned, so far as I am aware, have not yet been made public.

HEARTWATER

History.—The disease has been recognised in South Africa between forty and fifty years, and so far as is known it is peculiar to the country. By far the greater proportion of the country is free from the disease, it being found only in those localities where the bont tick (*Amblyomma hebræum*) exists. The districts affected are mostly the coastal ones, the Eastern Province of Cape Colony, the Transkeian territories, parts of Zululand, and the bush veld in the Transvaal, and Bechuanaland also harbours the disease.

Nature.—The disease is a specific febrile one, very similar in many respects to the plasmosis, but the causal organism belongs to the class known as ultra-visible, in that it cannot be seen with the aid of the highest power of the microscope, therefore its presence has to be presumed; but Lounsbury has clearly demonstrated that the disease is transmitted by a tick (the *Amblyomma hebræum*), and as it can also be transmitted by blood inoculation, the presumption that the disease is due to an organism or virus is apparently well founded.

The disease affects sheep, goats, and cattle, mostly calves, but a varying resistance is exhibited by the different breeds. Amongst the goats the Angora is the most susceptible, and in the sheep the Merino is more susceptible than the Africander, whilst the so-called Persian (which is really the black-headed Somaliland sheep) possesses a considerable degree of resistance, and although the animal can certainly be infected with the disease experimentally, yet it rarely contracts the disease in fatal form under natural conditions.

Symptoms.—The disease is ushered in by a rise in temperature which is high—from 104° to 108° F., 106° being fairly common. After this temperature has persisted for a day or longer, the ordinary symptoms of illness begin to appear: the affected animal will appear dull, lag behind the flock, and will remain quiet if left to itself. In many, if not a majority of, cases nervous symptoms present themselves at first in the form of nervous excitability and bleating, followed by fits or series of fits, in one of which the animal frequently expires. During a fit the eyes

are dilated, squint, or rotate in their sockets; the jaws may be closed, or there may be an in-and-out movement of the tongue or chewing movements of the jaws, twitching of the muscles, loss of control over the limbs; the animal falls to the ground with the head twisted to one side or back towards the withers; it moans and bleats piteously, and executes galloping movements with the legs.

In some cases the first intimation of the presence of the disease is the finding of an animal dead in the kraal or in the veld. The appetite is usually not entirely lost, although rumination may cease early in the attack, and the animal will generally feed a little.

The pulse and breathing are accelerated, and the exudation of the fluid into the heart sac, from which the disease takes its name, is denoted by a fall in the temperature, which drops below normal before death, and by uneasiness in the animal, manifested by lying down and getting up again. As the fluid increases in quantity the breathing becomes more laboured.

In sheep, when the disease is somewhat chronic in its progress, the wool becomes easily detached from the skin, and if the wool is grasped to restrain the animal it comes away in handfuls. The symptoms are most pronounced in goats; in cattle they are similar, but not so well marked.

Post-mortem Appearances.—On making a post-mortem examination, the most striking symptom is the presence of a clear, straw-coloured fluid in the heart sac. This may amount to as much as ½ pint, but is usually less; it is not invariably present, but it is usually so, hence the name given to the disease.

An exudation of this fluid frequently takes place into the chest cavity, less so into the abdominal cavity, and into the connective tissue adjacent to the larger veins, the neck being the site that is oftentimes infiltrated in this manner. This straw-coloured fluid is often tinged with blood, and a peculiarity exists with it when compared to dropsical effusions found in the same places, in that it coagulates quickly when exposed to the air, and when placed in a vessel it may do so in the space of a few minutes. The fourth stomach is congested, and a patchy congestion of the small intestines is often found, with the spleen slightly enlarged and the liver somewhat congested. The heart is usually pale, but otherwise normal. The lungs are pale, and the lobules may be slightly separated by the fluid.

Incubation.—The period that elapses between the inoculation of virulent blood until a fevered rise in temperature is shown is

from eight to ten days. The period of incubation after successful tick infestation in the laboratory is from eleven to eighteen days; after natural tick infestation on the veld, fourteen to twenty days.

Treatment.—Medicinal treatment up to the present has not proved successful, and despite a number of experiments an economic system of preventive inoculation for this disease still awaits discovery; therefore, as in the case of East Coast fever, the only effective method of suppressing the disease is to get rid of the ticks, and the best way to do this, so far as is known at present, is by a campaign of regular, systematic, and effective dipping of animals in an arsenical dipping solution of effective strength, and at sufficiently short intervals to accomplish the purpose. If this process of tick destruction had to be accomplished only by the means of dipping the sheep and goats on the farm, it would be an arduous one; but the bont tick is a greedy feeder, and will suck blood from most animals; therefore the big stock, particularly cattle, act as traps for conveying the tick to the dipping tank and destruction.

Practical experience has proved that by ridding land of ticks by the regular dipping of cattle the farming of small stock afterwards on such land is both possible and profitable, and a quantity of land has been, so to speak, reclaimed to economic farming practice by the dipping tank. This has been particularly noticeable in the coastal area of the East London district of the Cape Province, where large areas have been reclaimed for grazing sheep through the destruction of bont ticks, which has been effected by the enforcement of dipping of cattle for the prevention of East Coast fever.

PIRO- AND ANAPLASMOSIS

Preventive Inoculation in Cattle.

THE fact that recovered animals are immune under ordinary circumstances to further infection of redwater and gall-sickness, coupled with the necessity of taking cattle, particularly good well-bred ones, from areas where these diseases are not present to areas where they are enzootic, led to the idea of inoculating such susceptible cattle with these diseases by introducing the blood of an immune animal into their systems, and whilst the reaction set up by the disease was present keeping the inoculated cattle in a stable and free from ticks.

This method of inoculation was first practised to any extent in Texas, the procedure being to take blood from a young beast in good healthy condition that had been bred and reared in a redwater district, and that was presumed to have contracted this disease and recovered from it, and was therefore immune, and inject 5 c.c. of such blood into the susceptible animal. It was held, and I think correctly, that by limiting the amount of virus injected a milder attack of the disease was set up than by tick infection, and the results obtained justified the adoption of the inoculation on an extensive scale. The alternative to this method is to introduce susceptible cattle to the redwater area in the winter season of the year, when tick life is at its lowest ebb, and practical experience also appears to indicate that the disease transmitted by ticks at that season is less virulent than in the summer; but this may be due to fewer ticks being present, therefore a smaller quantity of virus is injected by them. This system of allowing imported cattle to acclimatise to enzootic tick-conveyed diseases is still largely practised in South Africa, and many importers, even although their cattle have been subjected to the preventive inoculation, only allow them to graze for the first time during the winter—a precaution that appears commendable by the results obtained.

Since the importation of pure-bred cattle from Europe has developed into a large trade, experience in the results obtained in the preventive inoculation of these animals, both in England

before shipment and in South Africa upon arrival, has led to a greater knowledge of these diseases being acquired. Amongst the most important matters brought to light was the fact that different strains of the disease existed, therefore it was advisable to inoculate the beast with the strain of the disease present in that part of the country to which the imported animal was going; and, further, as without testing it upon susceptible cattle one could not tell the virulence of the disease that the blood of any particular immune animal would convey, it led to animals having blood that conveyed the disease in a mild form being kept tick-free to prevent further infection, and being used simply for the purpose of supplying blood for preventive inoculation of cattle. Another important discovery was that the disease quickly became exalted by passage through susceptible animals; the blood from an immune animal might convey a mild form of the disease when injected into an imported animal, but if blood was taken from this imported animal and injected into other susceptible cattle it might, and probably would, set up a severe and fatal form of the disease. Further, a great difference in the susceptibility of cattle according to their breeding was discovered. With the native indigenous cattle of the country it was found that it was impossible to transmit the disease to calves until they had attained an age of six or eight months; the half-bred cattle from such native cows and an imported bull were less resistant, and were susceptible to the disease at about three months old, the susceptibility increasing as the breeding improved, until the pure-bred calf was liable to contract the disease even when only a few days old.

The individual susceptibility of cattle was also found to vary considerably. Broadly speaking, amongst the imported cattle it was found that those breeds and individuals that had led a natural life were more resistant and made better recoveries from the disease resulting from inoculation, and that breeds and individuals who from overfeeding, pampering, close breeding, or other causes that sapped the constitution, succumbed to its effects in greater numbers.

Age also plays an important part in the amount of resistance present; the younger the animal the more resistant it is, and with overseas imported cattle it has been found that up to fifteen months old may be considered a safe and desirable age for preventive inoculation, and the older the animal is after that age the greater is the risk attending inoculation. Therefore it will be gathered that now, when preventive inoculation is practised,

only blood of known virulence is employed; this is obtained from the different veterinary laboratories, the only drawback to its employment being that it has to be used while still fresh, within forty-eight hours after being drawn from the animal, and owing to the great distances between places in Africa, this makes its conveyance and use within the time in some cases a matter of difficulty.

The inoculation itself is a very simple subcutaneous one, the success depending to a great extent upon the after-treatment of the inoculated animals. It is most important that the inoculated animals should be kept absolutely tick-free during the time they are undergoing reaction, and it is advisable that they be not allowed to become grossly tick-infested soon after recovery. Of course, no cattle should be allowed to be grossly tick-infested at any time. Wetting has a deleterious effect upon the animal whilst it is undergoing reaction, therefore this should also be avoided.

The temperature of inoculated animals should be carefully watched and taken daily, and if the first reaction, which is the redwater reaction, and which takes place within fourteen days, threatens to be unduly severe (if the temperature rises above 104° F.), a dose of trypan blue should be given to control the reaction, the amount and manner of injection to be as recommended in the article on Bovine Piroplasmosis. This is particularly necessary in the case of well-bred animals. The only other treatment that is necessary is ordinary good management; the bowels should be watched and regulated by suitable food such as bran and green food, and careful nursing of any that may react severely to the disease.

Many owners who by regular dipping have eradicated these diseases from their farms now inoculate their young breeding stock before selling to owners in localities where these diseases are present.

It is customary when inoculating to use blood containing the virus of both redwater and gall-sickness, and by taking the temperatures of the animals daily to watch the reactions of the two diseases, that of redwater occurring at about the seventh to the tenth day and then subsiding, being followed in about three weeks by a second rise in temperature, denoting the reaction to gall-sickness. It has been found to frequently happen that in animals in which the reaction to redwater is severe the reaction to gall-sickness is mild in character, and in other animals it is vice versa.

SPIROCHÆTOSIS

Synonyms.—Spirilloses (French, *Les Spirilloses*).

Nature.—Includes a class of diseases due to the presence of a specific spirochæte in the system, the parasite varying according to the species of animal affected or the country in which the disease occurs. The spirochætes in structure more closely resemble the trypanosomes than the plasmodia; they are widely distributed through the world, and have been found in nearly all the domesticated and in several wild mammals, in birds, fishes, bees, and in a number of insects. A great number of the spirochætes are apparently harmless, but others, again, cause serious disease. Amongst the better known of them may be mentioned the well-known *S. obermeiri*, which is the cause of relapsing fever in man; *S. duttoni*, the cause of relapsing fever (tick fever) in man in Africa; *S. pallida*, the cause of syphilis in man. Veterinary knowledge is still to a great extent in the making in the differentiation of the disease-producing and non-producing spirochætes. They are transmitted by ticks, body lice, bed bugs, and probably by fleas.

History.—In South Africa Theiler discovered spirochætes in the ox, sheep, and horse in 1902. In 1904 he proved that they were transmitted by means of adults and larvæ of the blue tick, *Boophilus decoloratus*, and the red-legged tick, *Rhipicephalus evertsi*. In 1906 Dodd discovered a spirochæte in the pig. In 1905 Jowitt found a spirochæte in canker and grease in horses; he also investigated the disease in the fowl. This was first discovered in Rio Janeiro in 1903. Since these dates spirochætes have been met by other investigators. Bevan has frequently found them, and they are very prevalent amongst fowls in Rhodesia; he also often met them in calves suffering from what is known as " sweating sickness," a form of calf disease, and it is possible that eventually they may be found to be the cause of one or other of the diseases that at present little is known about.

The Disease in the Fowl.—At the present time this is the only known disease of this family coming within the scope of the veterinarian. It is transmitted by the tick *Argas persicus*, locally known as the " tampan," which tick is believed to have been introduced into South Africa from India. It is also found in Australia and New Zealand, and was probably introduced

there either from India or South Africa. It is found in dry climates, both hot and cold, and multiplies rapidly in them; it is not found in cold, damp climates, and is unknown in Great Britain. The disease affects fowls, turkeys, pigeons, and some wild birds, but principally fowls. It may occur at any season of the year, but is more prevalent in the spring months. In infected premises the fowls become immune to the disease, and the older birds survive the pest fairly well; but a number of the chickens succumb to it, and profitable poultry farming cannot be carried on when the disease is present. When susceptible fowls are introduced into infected premises they quickly contract the disease, in the majority of cases fatally, the incubation being about two days or longer. When such an introduction of new blood takes place it may not be only the recently imported susceptible birds that die; in some instances the disease, by passing through them, apparently increases in virulence, and in this state it is more than the immunity of the original stock can withstand, and many of them may also die. Fowls on infected premises also suffer from relapses of the disease; these may be due to the disease having somewhat increased in virulence by passage through susceptible chickens, or that the tampans have increased in number and the fowl-houses require spraying out.

Symptoms.—The symptoms in the acute type of the disease are high fever—109° to 110° F. (the normal temperature of the fowl is 106·5° F.)—and intense thirst, followed by abstinence from food, ruffling of the feathers, drooping of the wings, with a greenish-yellow diarrhœa. The bird becomes dull and disinclined to move; finally, weak and unable to stand, it falls down, the eyelids partially or entirely closed, death usually following in the course of a few hours, but it may occur quite suddenly.

In the chronic form of the disease, which frequently follows an acute attack, paralysis of some part of the body takes place, often in one or both legs. If the latter, the fowl finds great difficulty in walking, or cannot walk at all, and flutters along with its wings. If in one leg only, it will be seen that the bird cannot use its claws; they become doubled up as if cramped. In a small number of cases the neck may be the part affected, when the fowl's head may be completely twisted round, the bird continuously looking over its back. In a certain number of cases the symptoms gradually disappear and the birds recover; in other cases there is a gradual emaciation, and death follows.

Post-mortem Appearances.—The most pronounced symptom is the bloodless condition of the body; the muscles appear as if

boiled or macerated; the skin frequently shows small hæmorrhagic spots where the tampans have been attached.

Treatment.—The treatment to adopt is a preventive one, by ridding the premises and the poultry of the ticks. This is a job of some magnitude; it requires to be done very thoroughly, as the tampan is very tenacious of life, and can live without feeding probably up to two years. Adult ticks will travel considerable distances to get to poultry; therefore, when it is discovered that tampans are present, a thorough search has to be made to find all the places in which they have become secreted. The fowl-house should be either burned, pulled down, and re-erected, or else thoroughly sprayed with a solution that will destroy the ticks. Paraffin is perhaps the most effective agent for this purpose, used at a strength of 25 to 75 per cent. water; soap solution facilitates the mixing. It should be forced into all cracks and crevices by means of a strong spray pump or garden syringe. Trees and posts in the vicinity should be examined carefully to determine if they are harbouring tampans in or under the bark, or in cracks. After treating the fowl-house as suggested, it is a good plan to whitewash it out with some carbolic acid in the whitewash, not only for cleaning and sweetening the place, but it enables one to see the droppings of the tampans, very similar to fly-spots, near their cracks and crevices, and spots of blood from the fowls, if one's disinfection has not been thoroughly successful, which it is not likely to be.

The fowls should also be dipped at the same time to destroy the larval ticks that may be attached to their bodies. For this purpose hot water containing 5 per cent. of paraffin and soap to emulsify it is effective; Jeyes' fluid in a 10 per cent. solution is also suitable.

Such a dipping may also be used as a curative treatment. A bird that is seen drooping its wings or carrying one wing exactly as if it was broken will, if caught and examined, be frequently found to have larval ticks that appear as small blue-black spots attached to it. By destroying these by dipping and preventing it becoming reinfested by placing it in clean quarters gives the fowl a better chance of withstanding the ravages of the spirochæte in its blood.

In the case of valuable birds justifying medicinal treatment, $\frac{1}{15}$oxyl in 5 c.c. doses has been found a curative. Soamine, at grain in 1 c.c. of sterile water injected into the muscle, if used early in the disease, generally aborts the attack, and recovery takes place.

OTHER SKIN DISEASES

Senkoba Skin Disease.

This is at present an unclassified disease, and so far as investigation of it has gone, no mange or other parasite or specific organism has been found.

History.—The disease was first observed in Northern Rhodesia, where it got its name, about the year 1912. By 1915 it had become fairly prevalent, and in 1916 it had appeared in the Matetsi portion of the Wankie district, Southern Rhodesia. It broke out amongst cattle that had been imported from Northern Rhodesia, and these animals before importation remained for some time in the vicinity of Livingstone, a locality in which the disease was present.

Nature and Symptoms.—The disease is apparently confined entirely to the skin, any constitutional disturbance that may occur being due to the severity of the skin lesions. The disease in cattle usually commences on the bare skin of the body—that is, at such places as the armpits, groin, udder, and under the tail. The onset of the disease is characterised by an inflammatory condition of the skin, which dries and forms crusts that reach about ¼ inch in thickness at the places mentioned. These crusts may be continuous and extensive, covering the whole scrotum in males as well as the thighs and legs in an armour-like scab. In such cases the animal stands with its back arched, looking thoroughly miserable and repulsive, is disinclined to move, and if compelled to do so, owing to the cracking of the scab and the soreness of the underlying skin, the movement is attended with a certain amount of pain. On other parts of the body, particularly the back, isolated patches of scab about the size of a shilling piece occur, greatly resembling ringworm, except that the crusts are thicker and more firmly attached to the skin—in fact, quite an amount of force has to be used to remove them. After removal the skin under the crust is found to be raw, red in the centre, and moist with exudation. The head is usually the last part of the body in cattle to become affected with the disease.

In sheep and goats the symptoms of the disease are very similar to those in cattle, except that the head is the commonest site, the lips and ears being usually badly affected. The scabs on the lips assume a papilliform shape and look like so many warts, giving the unfortunate creature a loathsome appearance.

Method of Infection.—Both in Northern and Southern Rhodesia the tick *Amblyomma variegatum* is accused as being the cause of the disease.

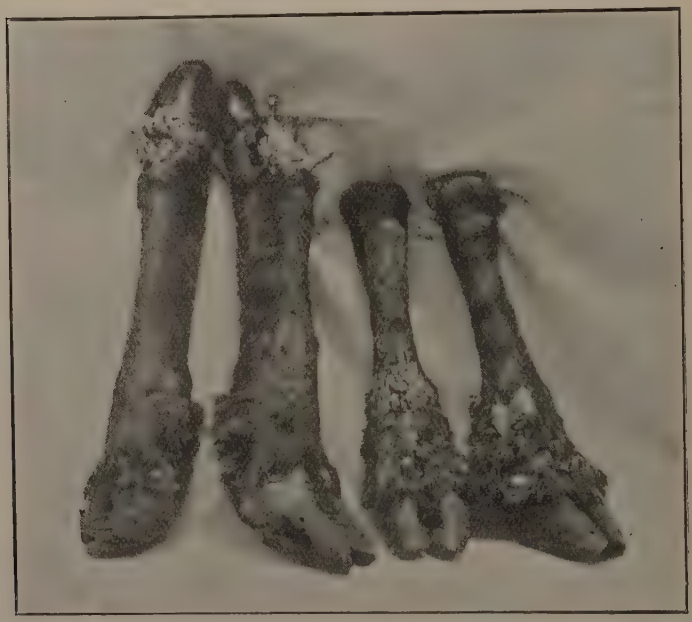


Fig. 14.—Senkoba Skin Disease.

This tick, so far as could be gathered, was new to the Wankie district, none of the European residents or bushmen living in the district having seen it before; and as this tick is easily distinguished by its markings—in fact, they are conspicuous—there is little chance that so keen observers as the aborigines could have overlooked it. It may be, I think, safely assumed that this tick was new to the locality, and was introduced there by means of the before-mentioned cattle.

The tick is unknown in Southern Rhodesia, except on quite the other side of the country, in the Umtali district, where it was accused in the early years of the present century of transmitting

a certain form of epizootic lymphangitis in equines; in other countries where it exists this tick is also held responsible for skin diseases. In the Matetsi area the tick was not found in great numbers; twelve adults on one beast would be the most that were ever found, and usually only one or two adults were present. The sites are those where the skin is thin and with few hairs—in other words, the same places where the disease first manifests itself. The presence of these ticks upon a beast is denoted by a primary swelling about the size of the end of the thumb arising at the spot where the tick has become attached.

If *Amblyomma variegatum* is responsible for this disease, which apparently is the case, there is also another factor necessary, because cattle in some herds had the tick upon them and yet the disease did not develop, but the disease has only been seen in herds where *A. variegatum* was present.

Treatment.—Several animals that became badly affected, practically covered all over with crusts, succumbed to the disease; others as badly affected responded to a treatment of hand-dressing with paraffin. Loosening the scab by washing with soft-soap and warm water and then applying a lime and sulphur dip effected a cure in a number of cases, but the treatment indicated undoubtedly is a preventive one, and experience has taught that this is found in regular weekly dipping in the ordinary arsenical dips, which not only prevents the disease appearing, but is also a curative to a considerable extent, many affected animals having been cured by this means.

SCABIES, OR SCAB

Scab in South Africa is usually regarded as denoting a disease in either sheep or goats; when it occurs in other animals it is called mange, and in man it is known as itch. Although the disease may be variously named according to the animal and part affected, it is caused by what are known as mange mites, which cause a contagious disease of the skin manifested by intense itching and an eczematous inflammation.

The mange parasites are very small mites belonging to the order of the Acarina, divided into classes according to their method of life or structural peculiarities, and the animal upon which they live. All animals, including man, apparently have at least one variety of mange mite that preys upon them. Some animals have several varieties parasitic to them; likewise some varieties of mange mites can live and thrive upon more than one species of animal, whilst others are peculiar to their particular host.

The classification of the mange mites is not always arranged in the same order, particularly by English and Continental authorities, one authority using a different classification and nomenclature to another, making a comparison of them rather confusing. But for practical purposes they may be divided into two broad classes of—

1. Parasites which live on the surface of the skin, technically designated Dermatocoptes, which include Psoroptes and Dermatodectes.

2. Parasites which penetrate into the skin glands and hair follicles, designated Sarcoptes, which are again divided up.

These parasites give rise to many forms of the disease of mange, some of them rarely met with in any country, others that have never been diagnosed as existing in South Africa; therefore, in dealing with them only those forms that have been met with, or are likely to be met with, in South Africa will be described.

With regard to naming the parasite that causes the particular form of mange, I quote the following as an example of a case where authorities differ. Wallis Hoare, in his book on " Veteri-

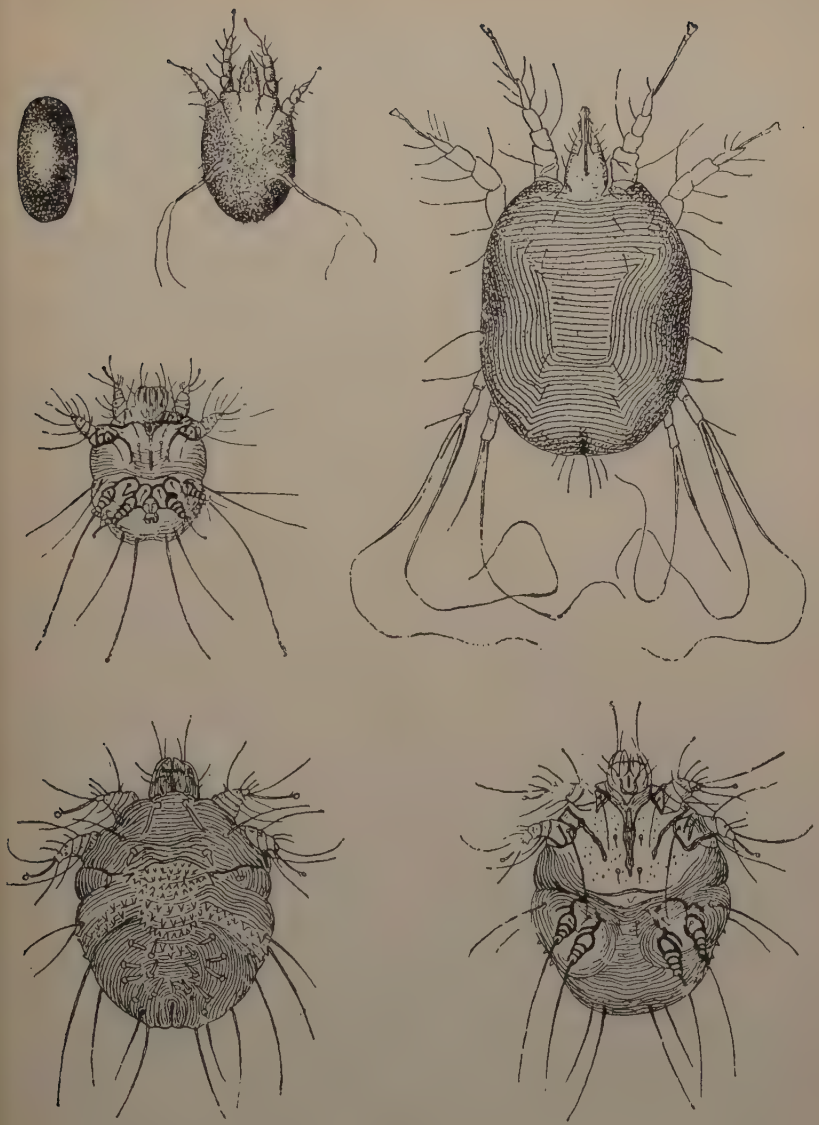


FIG. 15.

Left top figure, egg of mite which causes common sheep scab; middle top figure, six-legged stage of sheep scab mite; right top figure, young female before moulting for the last time, dorsal view; middle figure, adult male parasite of sarcoptic scabies of man (the corresponding parasite of sheep is very similar), ventral view, × 250 (after Blanchard); left bottom figure, adult female parasite of sarcoptic scabies, dorsal view, × 250 (after Blanchard); right bottom figure, same, ventral view (after Blanchard). All greatly enlarged. (From Moussu and Dollar.)

nary Medicine," when dealing with sarcoptic mange of the horse, states: " Psoroptic is the usual form of mange met with on the horse's body. It is due to the *Psoroptes communis equi*. Probably 95 per cent. of the reported cases belong to this type." Mohler and Eichhorn, in their translation of Hutyra and Marek's book, state: " In the great majority of cases scab (mange) in the horse is caused by the *Sarcoptes scabiei*, far less often by *Dematocoptes* (*psoroptes*) *communis var. equi*."

Bedford (Pretoria), in his report of 1915, states under *Psoroptes communis var. equi* : " I know of no record of this variety having been found in South Africa."

Therefore, in naming the parasite that causes scab or mange in South Africa, I shall only name such as have been determined by scientists within the country.

MANGE

Psoroptic and Sarcoptic Mange in Equines.

(NOTE.—When it is proclaimed that mange in horses, mules, donkeys, and camels is a notifiable disease, I take it to mean that only the commoner forms, the psoroptic and sarcoptic, are meant.)

Synonyms.—Itch, Sweet Itch, Riff, Duke, Fiddle, Scabies, and Scab.

Nature.—Mange is a contagious skin disease caused by mange mites, and manifests itself by intense itching and by a more or less violent eczematous inflammation of the skin. It is frequently difficult to account for the manner by which an animal has contracted mange; it may have never been in contact with another animal, and the missing link is that the life history of the mite, when it is apart from its host, is not known. We know that a dirty ungroomed condition of the body is a predisposing cause, the same as it is with lice both in animals and in man, but no amount of dirt by itself can lead to the production of a living insect or any other living matter; life can only spring from pre-existing life. We know of many ways by which the disease spreads from animal to animal, and that it has been estimated that in three months a pair of insects can give rise to one and a half million descendants, but isolated outbreaks of the disease occur that cannot be satisfactorily accounted for. During the Great War the mange mite was found on rats; this may account for a certain number of mysterious outbreaks. The important point to remember in connection with the treatment of the disease is the time occupied in the different stages of the mite upon the host. The adult female lays eggs which hatch out into larvæ in from four to six days; the larvæ take from eight to ten days to become adults, at which stage they, of course, can lay another crop of eggs; and the treatment to be aimed at is to destroy the mites before they can lay eggs, as mites are easier to destroy than the eggs are. (NOTE.—The foregoing are times given by scientists working in Europe. Perhaps when

investigations are made in South Africa the same difference as in sheep scab may be found.)

Symptoms.—The earliest symptoms will be rubbing, biting, kicking, and general signs of itchiness; the hair upon the affected areas may become stained and wet from the effects of the teeth. When the disease is present in a stud of animals, a well-known method of detecting its existence in the early stages before the symptoms have become marked is to rub the animal on the withers with the tips of the fingers, when, if it is affected, it will usually stretch out the neck and move the lips quickly, expressing a pleasant sensation. I have heard this movement of the lips described as the horse laughing.

On examining closely the affected portion of the skin, papules and vesicles will be seen which afterwards become bare patches. By increasing in number and forming together, large hairless areas are formed. The bare places are at first scaly, but if the case is not attended to and allowed to take its course, the scales become thick scabs, and the skin becomes thickened and wrinkled, and looks very much like an elephant's hide. When a case has reached this stage the irritation is frequently not so great, and the animal is usually greatly emaciated; but earlier in the disease the irritation is often intense, and it is no uncommon thing to find animals with extensive, almost raw abrasions over the body, caused by gnawing and rubbing.

Diagnosis.—Psoroptic mange usually commences on those parts of the body covered with the strongest hairs, such as the withers, sides of neck, back, and root of tail. Sarcoptic mange favours those portions of the body where the skin is thin, as the belly and brisket, also between the jaw bones. This is the form of the disease that commonly affects camels, but as South Africa does not at the present time possess any number of camels, the matter is not of great importance. Naturally the site either of these diseases starts at depends greatly upon the way they have been contracted. If they have been conveyed by the harness, the primary lesions will be found where the harness has been in contact with the body.

Psoroptes can be seen by the naked eye if a scraping from the affected part is put on black paper and slightly warmed, and whilst it may be possible to differentiate between these diseases (psoroptic and sarcoptic mange) in some cases, positive diagnosis can only be obtained by examining the mite under the microscope. In fact, it is held that mange in equines can only be correctly diagnosed by this means for the reason that the mites

found in forage and hay dust frequently get upon horses, inducing itchiness, rubbing, and other symptoms simulating mange; further, these mites are very similar in structure to the mange mites, and can only be distinguished from the latter by a careful examination.

Treatment.—Treatment of mange, to be successful, must be thorough—in fact, probably more depends upon the thoroughness of the application all over the animal than upon the remedy used. In the first place, the animal should be clipped all over, the legs and mane included, before remedial treatment is attempted, and, owing to the itchiness of the patient, this is not always an easy proceeding. A dressing that I have been successful with is as follows:

Paraffin	1 pint.
Soap solution	1 gallon.

Shred 1 pound of yellow or Sunlight soap into 1 gallon of warm water, and keep this stirred and simmering over a slow fire until the soap is dissolved; then remove from the fire, add the paraffin, and stir until the two are mixed into an emulsion of creamy consistency. Rub this well into every part of the animal's body. On the sixth or seventh day wash animal thoroughly with soap and warm water, and after drying again apply the paraffin emulsion; in another week repeat the washing and dressing, when as a rule the animal is cured.

Another and perhaps more effective method is to adopt the foregoing treatment, but on the day following the application of the paraffin emulsion to rub well into the skin an ointment composed of 1 part of sulphur and 4 parts of lard.

A dressing suggested by the Veterinary Department of the Board of Agriculture in England is as follows: Lime, powdered sulphur, and water in the proportions indicated below. A convenient quantity for a large establishment to make up at a time would be 9 pounds of lime and 18 pounds of sulphur. Slake the lime and make into a thick paste with the sulphur. Place the mixture in a strong cloth, tie the ends, and suspend in a boiler containing 10 gallons of water, so that the water completely covers the contents of the cloth. The cloth must not touch the sides or bottom of the boiler, as otherwise the cloth may be burnt and its contents escape. Boil for two hours, then remove the cloth, taking care not to break it when doing so, and throw the solids away. Make up to 10 gallons again with additional water, and put the liquid into a tight drum or barrel.

Application.—For use as a preventive, dilute the fluid with ten times the amount of water—*i.e.*, 1½ pints of the fluid to every 2 gallons of water—and apply with a spray to all parts of the horse's body.

For affected horses, dilute the fluid with eight times the amount of water—*i.e.*, 2 pints of the fluid to every 2 gallons of water—and apply with a spray to all parts of the horse's body.

Fig. 16.—Tick Destruction (Native Dipping Tank of Stone).

Quantity Used.—Two gallons of the diluted fluid is sufficient to treat one large horse. It is essential that every part of the body and legs be well drenched with the spray.

In South Africa cattle-dipping tanks are usually within easy distance of almost everyone, and the treatment of mange can be simplified considerably by dipping affected animals weekly in the arsenical solutions of the ordinary strength used for destroying ticks.

Prevention of a recurrence of the disease must be aimed at by isolation of infected animals, and certainty that all mites have been destroyed on their bodies before they are allowed to mix with other animals; the hair clipped off should be burnt; all

harness, brushes, stable posts, etc., that the animals may have rubbed against or otherwise come in contact with should be thoroughly disinfected.

During the Great War, in the British Army in France the practice of dipping horses in arsenical fluids was found to be the most effective of all treatments for the cure of the many cases of mange that occurred.

The method was as follows: The animals were first clipped all over, and were then immersed every three days in a dipping-tank

Fig. 17.—Tick Destruction (Native Dipping Tank of Stone).

in a solution of arsenical cattle-dip at what is known in South Africa as the "three-day strength." Watkins Pitchford of Natal was the discoverer of this treatment (see section on East Coast Fever), and his prescription for the "three-day dip" is as follows:

"Four pounds arsenite of soda (80 per cent. arsenic), 3 pounds soft-soap, 1 gallon paraffin, 400 gallons of water, prepared as follows: Dissolve the soap and arsenite separately in a sufficient quantity of hot water; add the soap solution to the paraffin and beat into an emulsion; then add water to make up to 400 gallons, stirring vigorously the while."

Several proprietary arsenical cattle dips are now in the market in South Africa and elsewhere; these all contain instructions for the amount of water to be added to prepare a three-day dip.

In France the dip used was Cooper's Improved Cattle Dip, in the strength of 1 gallon of dip to 300 gallons of water, which is the original strength this dip was used as a three-day cattle dip, and is slightly weaker than the three-day cattle dip at present in use. There is no reason to suppose that horses would not tolerate the stronger three-day solution equally with cattle; in fact, the latter animals, when they have become accustomed to dipping, can tolerate a considerably stronger solution.

The result obtained amongst the army horses was, when compared with other methods of treatment, most satisfactory, owing to its simplicity and easy application, little difficulty being experienced in inducing the animals to enter the dipping-tank on the first occasion, and afterwards no difficulty at all.

The majority of cases were cured in a month or slightly under, but occasionally an animal would be found that had a recrudescence of the disease in a small spot, which, if overlooked or neglected, would again spread over the body.

Sarcoptic Mange in Cattle.

This disease is not of infrequent occurrence in different parts of the world, particularly in stabled cattle, and is spread to a great extent by brushes and cloths. The parts most frequently affected are the root of the tail, the groin, and mammary gland. The disease does not spread with any great rapidity, but if neglected it will extend to other parts of the body.

The symptoms are similar to other manges; there is itchiness and rubbing of the affected part, which becomes hairless, covered with scab, and the skin corrugates and thickens.

Diagnosis demands the demonstration of the mite in the yellow liquid obtained at a fair depth in the lesions under the microscope.

Treatment.—This can be based on those recommended for mange in the horse.

Sarcoptic Mange in the Pig.

The parts most frequently affected are the ear, around the eyes, the withers, the armpits, and inside the thighs. The itch is intense, and the animal lacerates the skin by scratching with

the hind feet and rubbing the body against objects. The skin becomes reddened and the hair falls out; later on it thickens and wrinkles. The sarcopt in the pig can often be seen with the naked eye in scrapings placed in a glass tube.

Treatment.—This is the same as in the horse, but owing to the thickness of the skin in pigs it is advisable to soften it as much as possible with the sulphur and lard ointment, and use the scrubbing-brush to apply paraffin emulsion. Pigs can be dipped in the ordinary arsenical cattle dips.

Sarcoptic Mange in the Dog.

This disease frequently starts on the head, and by means of the paws is transplanted on other parts of the body, including the paws; other common sites are the chest, elbows, and abdomen. Actual diagnosis is a matter of difficulty, owing to frequent inability to demonstrate the parasite. Irritation is always great, the animal biting and scratching the affected parts.

Treatment.—Dressing the affected parts with sulphur ointment, and baths in one or other of the popular coal-tar disinfectants such as Jeyes', usually cures the disease. Another prescription is creolin 1 part, soft-soap 1 part, spirit 8 parts. If the disease covers a great extent of the animal, it is not advisable to dress the whole body at one time, but only a portion—about a quarter. The habit of dogs to lick and bite the body must be remembered when applying remedies to the skin, as it is possible to poison the animal by this means.

Sarcoptic Mange in the Cat.

This disease in the cat is often known as notœdric mange. The symptoms are the same as in other animals, but in my experience it is a very severe disease. Kittens and many full-grown cats die from its effects, and it spreads to dogs; but perhaps its worst feature is that it is contracted by man, and causes a disease (mange) most difficult to cure. Great care should be taken to prevent children becoming infected by coming in contact with affected cats, or with any place or thing that has been contaminated by such animal.

Chorioptic or Symbiotic Mange in the Horse.

This is a common form of mange that affects the limbs of horses, causing the animals to kick and stamp their feet on the ground,

severely jarring, if not injuring, the limb; rubbing one hind leg against the other is a typical symptom. The disease is more frequently seen in the hairy-legged and commoner types of horses and in animals that are not well-groomed.

Treatment.—This consists in clipping the limbs, well washing with soft-soap and water, and then applying one or other of the mange dressings. In three weeks a cure should be effected.

SCAB IN SHEEP

THE disease that is universally recognised as scab in sheep is caused by the mite known as *Psoroptes communis* var. *ovis*, a surface feeder.

History.—Sheep scab is not a new disease of obscure origin and mysterious nature. On the contrary, the historian may trace its existence back to the earliest ages—may, indeed, as a starting-point refer to Leviticus xxxii. 22, in which the offering of a "scabbed sheep" is prohibited; may ramble on through the classics of Greece and Rome to prove that the ancients were familiar with the malady, and did not underrate its virulence; and then follow its history right up to the present, when its ravages are still costing South Africa a very heavy sum of money annually through the loss of wool and animals.

Nature and Symptoms.—The *Psoroptes communis* var. *ovis*, the mite responsible for the disease, is something like a crab in appearance; it is about $\frac{1}{50}$ inch long and about $\frac{1}{80}$ inch broad; therefore it is visible to the naked eye of those possessing good eyesight. It may be demonstrated by shaking a piece of scabby wool on to black paper, and examining the débris through a pocket lens. The mites invade the parts of the body most covered with wool. In advanced cases the disease will be found on the neck, shoulders, withers, back, sides, and flanks, and sometimes on the breast also; but as a rule the mites keep away from the under surface of the body, and distinctly avoid those parts where wool is sparsely grown or altogether absent.

The disease is one of debilitated sheep that makes its appearance with greater frequency when food is scarce and innutritious; it may disappear without any treatment when food again becomes plentiful. This habit of the disease has been noted for a long time in South Africa, and the popular theory that the mites cannot thrive and increase and multiply in animals in good condition that have an amount of fat in their skin and wool has been corroborated to a great extent by the experiments of Shilston in Natal.

The symptoms of the disease are simple. In early stages attention is drawn to an injured fleece; it appears as if one had

used a pair of horse-clippers and clipped a patch of wool away, leaving it attached and hanging from the sound wool. The fleece will be noticed to be brittle, and can be pulled from the skin in patches. When an affected sheep is scratched with the fingers, it turns up its upper lip and champs the teeth in token of appreciation of the sensation produced. If the skin is examined where a patch of wool has become detached, small spots about the size of a match-head will be seen, yellowish in colour, and caused by the attacks of the parasites. These spots become

FIG. 18.—AN ADVANCED CASE OF COMMON SCAB. (From Moussu and Dollar.)

larger and burst, the discharge forming a crust under which numerous parasites may be found.

Treatment.—Dipping, pouring, and salving are methods of treatment that have from time immemorial constituted an essential part of the routine of sheep farming. Hundreds of tons of mercurial ointment have been made for the special purpose of salving scabby sheep; arsenic in amounts that cannot be estimated, sulphur, tobacco, and carbolic acid have all been employed as the active agents in "dips" for the cure of the disease; and, according to observation, most of the preparations which have been long and now are offered as specifics for sheep scab are effective.

Some few years ago a controversy, which extended over a considerable space of time, took place in the South African agricultural press on the relative efficacy of various "dips" for not only curing the disease, but also the effect on the wool. As before stated, most of the dips are effective, but with the exception of the lime and sulphur dip prepared as directed by the Sheep Division in the Union, all of them failed to prevent the hatching of the eggs of the mite when tried by Shilston, who remarks: "The value of this property of the lime and sulphur dip cannot well be over-estimated, and it may partly account for the success attained in the eradication of scab from Australia; this dip was largely employed in effecting that happy result."

To Prepare Lime and Sulphur Dip.—Take 25 pounds of well-sifted flowers of sulphur and 20 pounds of slaked lime or 15 pounds of unslaked lime (this latter being preferable), add 3 or 4 gallons of water to the lime, and mix thoroughly with the sulphur into a paste. Throw the mixture into a pot containing 25 to 30 gallons of boiling water, and allow this to remain boiling for thirty to forty minutes, until a dark purple scum rises to the surface. The liquid (with the sediment) should then be placed in a tub with a bunghole 4 inches from the bottom, and after the sediment has settled the pure fluid may be run off into the dipping tank and the refuse thrown away. To every 25 or 30 gallons of dip prepared in the above manner should be added 70 or 75 gallons of clean water, to bring the mixture up to 100 gallons in respect of every 25 pounds sulphur and 15 or 20 pounds of lime used. It is desirable that the temperature of the liquid in the dipping tank should be maintained at a lukewarm heat throughout the operation of dipping, and in order to maintain this it is advisable that an additional pot or vessel be kept for hot water. In order to obtain good results when using this dip, it is essential that the lime should be of good quality and be freshly and thoroughly burned and slaked only when it is required for dipping purposes. In this state the lime consists of almost pure calcic oxide, but on exposure to the air for any time this calcic oxide (essential for the thorough dissolving of the sulphur) becomes changed into calcic carbonate, which latter does not combine with the sulphur to form the soluble sulphides required.

A study of the life history of the scab mite is necessary as a help in determining the intervals between and the number of dippings required. This has been done in South Africa by Shilston and Bedford, and their findings do not entirely agree

with those of Gerlach, who is the usual authority quoted for European conditions; but as Shilston worked in Natal and Bedford at Pretoria, and at different seasons of the year, their conclusions must be accepted as the conditions pertaining in South Africa.

The female mite lays up to ninety eggs on the skin. From these eggs a six-legged larva is hatched. The larvæ change their skin and become nymphæ with eight legs; these in turn again moult and become the mature male and female insects. Mating takes place and eggs are laid, completing the cycle, which is accomplished in nine or ten days. Gerlach states fourteen or fifteen days are occupied by the cycle, a difference in time that greatly affects the rapidity with which the mites may increase in numbers and intervals to be allowed between dippings in eradication of the disease.

The interval between dippings usually advised is fourteen to eighteen days, but in South Africa the time adopted has been twelve to sixteen days. Shilston advises an interval of ten days between the first and second dipping, which is usually sufficient, if thoroughly carried out, to effect a cure; but to greatly increase the chances of a complete cure, he suggests a third dipping at an interval of ten or fourteen days after the second.

With regard to the number of dippings required, sometimes a single dipping will effect a cure, but in the majority of cases this is not so, and its failure is usually attributed to the inability of the dipping fluid to destroy the vitality of the eggs—a belief that is apparently well founded; therefore it is customary to dip a second time. In badly infected cases there is a possibility of even a second dipping, according to the dip used, not being able to reach some of the mites that may be under hard crusts, and which may cause a recrudescence of the disease weeks later; in such cases a third dipping is advisable.

There is a belief widely held that places in which scabby sheep have been enclosed, such as kraals, stables, etc., harbour the disease for many months, and are capable of infecting sheep if put into them. This belief appears to be erroneous, as Shilston and other investigators, after numerous experiments, have arrived at the conclusion that neither the eggs nor the mites can live apart from the host for more than two or three weeks, and therefore that kraals, etc., can be used without fear of infection after one or two months' quarantine.

Scab in Non-Woolled Sheep and Goats.

The foregoing refers to scab in woolled sheep. Scab in non-woolled sheep and goats in this country is generally caused by parasites which burrow under the skin (sarcoptes). The first symptom is intense itching. It attacks the trunk, udder, belly, and limbs. If neglected it becomes generalised very rapidly; the animal wastes, and the mortality is great. Little pustules are found discharging a viscous liquid which forms crusts. The animals rub themselves raw, and the parts become dry and bran-like scales appear. After a time the hair falls out and the patch becomes larger and wrinkled.

The treatment and cure of scab in non-woolled sheep to a farmer possessing a cattle-dipping tank is very simple. The arsenical dips used for the destruction of ticks on cattle are efficient remedies for the disease. All that has to be done is simply to put the sheep or goats through the ordinary cattle dip every week until the desired effect is obtained. Cases can be quoted where farmers have dipped their sheep in this way for months—in fact, some farmers make it a regular practice to dip their sheep weekly to destroy ticks, and as a preventive of disease; and before the time that the parasites can live off the host had been scientifically determined practical experience had demonstrated that weekly dipping cleaned the kraals as well as the animals.

Ear Scab in Goats.

According to Shilston and Bedford, the above disease is very widely distributed in South Africa, and is caused by the *Psoroptes communis* var. *capræ*. The parasites confine themselves to the interior of the ear, and attempts to infect goats on the body with this parasite have failed.

They do not appear to set up the intense irritation evident in sheep scab, but infected animals frequently shake their head and scratch the back of the ears with the hind feet.

The treatment is said to be simple, 1 part of petroleum to 2 parts of cocoanut oil being an effective dressing; one application is sufficient to destroy both the mites and their eggs.

FOLLICULAR MANGE

FOLLICULAR mange is caused by the *Demodex folliculorum*, a microscopic parasite that takes up its abode in the sebaceous glands and hair follicles, and gives rise to the disease.

The parasite has been found upon man, the dog, cat, goat, pig, sheep, ox, and horse. In man the demodex may be found in many positions without apparently causing any harm, and with the exception of the dog, disease caused by the organism is rarely met with in the other animals.

In the dog the disease is of frequent occurrence, and is met with in nearly all parts of the world. It is commonly known as red mange or demodetic mange, and is a most intractable disease to treat. It is regarded as a contagious disease, and, considering its cause, it could not be regarded otherwise; but practical experience provides a great number of instances where the disease has failed to spread by cohabitation; furthermore, it is a difficult matter to establish the disease by artificial infection. Failures to do so by direct inoculation have been recorded in many instances, all of which goes to show that certain predisposing conditions, which are unknown, are essential before the disease can be established.

Treatment.—The disease is a difficult one to cure, and the treatment has usually to be carried on over an extended period. Cures are effected, and specifics are claimed to cure the disease with few applications. When such cures actually take place, in great probability the disease that has been treated has not been follicular mange, but something simulating it, perhaps eczema. The disease should be diagnosed by the demonstration of the demodex under the microscope.

The following treatments have at times been successful in effecting a cure. In localised cases of the disease clip the hair very short around the infected area and apply the following treatment: Creosote 1 part, liquor potassæ 2 parts, and olive oil 14 parts. Another treatment which I have found successful is to give the dog daily a bath in sulphuret of lime for one month, followed by a month's treatment of three baths a week.

The latest treatment, and one that promises better results and is worthy of further investigation, is autogenous vaccination.

Follicular Mange in Cattle.

In Europe the disease has been observed in herds of cattle but rarely; more frequently in North America; and, according to Probst, in German East Africa it is of frequent occurrence and enzootic in distribution, and is said by him to be there regarded as harmless, an opinion that is evidently not shared by the Veterinary Department of the adjoining country of Northern Rhodesia, where this disease also exists, and whose Administration issued a pamphlet on the skin diseases of cattle in that country, from which I extract the following: " The skin diseases of cattle in this country (Northern Rhodesia) may be roughly divided into those of parasitic and non-parasitic origin. The chief parasitic variety is follicular mange."

Symptoms in Cattle.—Usually the first symptoms take the form of nodules, about the size of a maize grain, under the skin. This condition is most commonly noticed on the shoulders and down the sides of the neck, but may occur on any part of the skin. In this stage the disease is often not recognised by laymen, or, if their attention is drawn to it, they attribute the condition to fly-bites or other comparatively harmless causes. The disease is slow in progressing, especially if the cattle are regularly dipped.

During the next stage these nodules increase in number, until in many cases they practically cover the whole under surface of the skin; they finally burst spontaneously and discharge a creamy, pus-like material.

In a large proportion at the later stages one finds patches of skin, the size of a half-crown piece or larger, denuded of hair and of a grey colour, and the skin in the neighbourhood of the dewlap, shoulders, and around the eyes becomes corrugated and very unsightly.

The disease is contagious; the causal parasite may be transmitted directly from one animal to another or indirectly through the medium of kraal posts, etc. Contagion may take place between different species of animals.

The character of the eruption and the course of the disease vary a great deal.

The disease in many cases has been found to remain practically

stationary in the primary nodular form for at least eighteen months, if not longer.

The debilitating conditions evidently play a part in accelerating

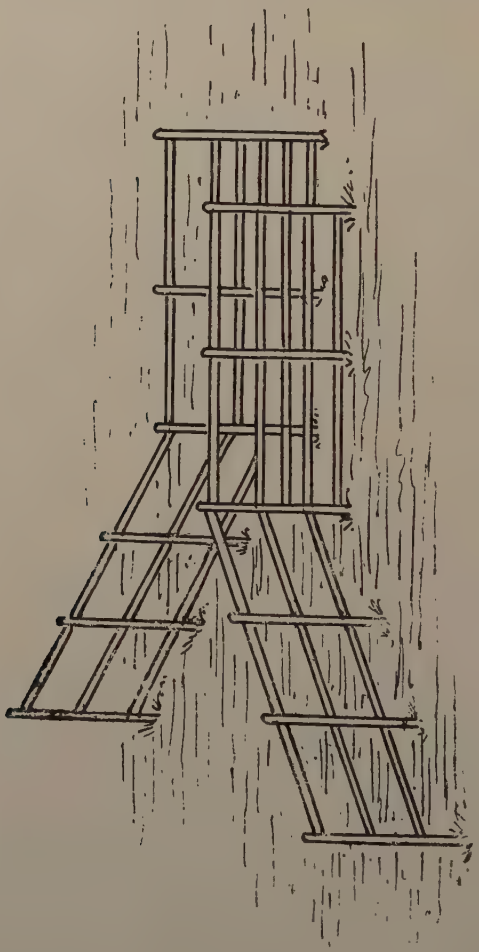


Fig. 19.—Cattle Race.

the course of the disease, particularly tick infections (infestation ?) and scarcity of food, as in the dry season in this country.

Treatment.—Preventive measures are more important than curative treatment.

New purchases should be carefully overhauled for the least

sign of the nodular eruption under the skin, and, as far as practicable, clean cattle should be kept away from other herds.

Regular dipping in dips of known strengths, disinfection of, and cleanliness with regard to, the cattle kraals, all tend to prevent an outbreak.

If follicular mange is already present in a herd, immediately separate the clean from the unclean, and proceed to destroy all advanced cases, burning the skins of animals thus destroyed.

The milder cases should be strictly segregated (herded in separate kraals by separate herds) and dipped weekly in Cooper's dipping fluid (1 in 200). (NOTE.—Presumably any other arsenical dip would serve the same purpose.) In the majority of affected herds there are only a few really bad cases, although very frequently a great number of mild ones.

Curative measures take quite a secondary place. They are always dangerous, unless strict isolation can be observed. It is advisable only when there are many quite moderate cases, which may be prevented from becoming too bad to work or cured sufficiently to render them fit to slaughter for native consumption.

Advanced cases are incurable, and in severe infection, where apparently a cure has been effected, a relapse inevitably occurs after a few weeks or months. Mild cases may be cured, but it is doubtful whether even these can be so cured that a relapse is impossible.

SECTION II

DISEASES CAUSED BY INSECTS

THE insects are air-breathing and possessing a windpipe, with the body divided into three distinct parts: the head, thorax, and abdomen. They possess three pairs of legs, and most frequently two pairs of wings. There are many orders, only two of which, Hemiptera and Diptera, contain parasitic species.

HEMIPTERA

The hemiptera is an order of insects the individuals of which generally have two pairs of wings. The mouth parts are fitted for sucking, and the metamorphosis is incomplete. It contains two families of interest to the veterinarian—the bugs (Cimicidæ) and the lice (Anoplura).

Metamorphosis.—By this term is meant a marked change in the structure of an insect in its development from the newly-hatched individual. In the order Anoplura (lice) the form which hatches from the egg is similar to the sexually mature form or adult, except in size. Such an insect is said to be without metamorphosis. Other forms are similar to the adult, except in size and the absence of wings. The wings slowly develop as the insect grows and moults its skin. Such a form is said to have incomplete metamorphosis. Flies, butterflies, bees, etc., hatch from the egg, and are in form much like a worm. This form is known as the larva. After feeding and becoming full-grown this larva passes into the resting or pupal stage, and is known as a pupa or chrysalis. From this pupal form the adult emerges. Such an insect is said to have complete metamorphosis.

THE BUGS (**Cimicidæ**).—The best known of these is *Cimex lectularius*, the bed bug of man. There is another bug that attacks poultry and causes the hen to desert her eggs.

Phthiriasis (Lousiness, or Pedicular Disease).

There are a great number of lice affecting different animals. Those infecting man are known as Pediculidæ, and include *Pediculus capitis*, the head louse; *P. corporis* or *vestimenti*, the body or clothes louse; and *Phthirius pubis*, the pubic or crab louse. Those affecting the domesticated animals, of which there is apparently at least one or more varieties parasitic upon each class of animal, are known as Hæmatopinus. There are apparently a goodly number of species in South Africa, of which the following have been determined: *Hæmatopinus macrocephalus*, or horse louse, found frequently at the base of the tail and in the mane; *H. eurystirnus*, or ox louse, found frequently in the ears of cattle; *H. stenopsis*, or blue goat louse, a very common one, especially affecting kids; *H. urius*, or hog louse, which kills young pigs.

Treatment.—Treatment in the larger animals is facilitated by clipping, and the "nits," the eggs of the lice, that may not be removed by the clipping will be to a great extent destroyed if the animal is singed. The treatment that is easiest applied in South Africa and that is most efficacious is dipping the animals in the ordinary arsenical dips of the cattle tanks. Of course, such treatment is not in all cases available or applicable to all classes of animals; therefore other methods must be considered.

One of the most efficacious is a freshly-made decoction of tobacco placed in a bottle with a quill through the cork, which simplifies application. Many of the proprietary dips in the strengths recommended can be used in a similar manner. Jeyes' fluid as a wash is also effective; vinegar is recommended, particularly for lice on calves. Mercurial ointment is a certain cure, but it must not be applied over large surfaces of the body at one time, for fear of mercurial poisoning, and is dangerous to use on dogs for this reason, unless they are securely muzzled.

For pigs, oil is the best treatment, particularly linseed oil applied to the skin.

For poultry, dipping in a solution of Jeyes' fluid, afterwards dusting insecticide powder (pyrethrum) into the feathers.

The preventive treatment consists in keeping the body and the living quarters of the animal as clean as possible.

DIPTERA.

Diptera is an order of insects, the members of which have only one pair of wings and a pair of halteres (balancers). In certain parasitic forms such as fleas the wings and the halteres are lacking. The mouth parts are fitted for sucking. The metamorphosis is complete. This order contains three important suborders.

1. *Nematocera.*—This comprises the gnats and mosquitoes, which, so far, are only suspected of transmitting animal diseases; and the sand flies (Simuliidæ), which attack fowls in South Africa.

2. *Brachycera* (*Flies*).—This includes Tabanidæ, Hippoboscidæ, Asilidæ (hornets), and Muscidæ (house fly, tsetse, and stomoxys). These are described farther on.

3. *Aphaniptera* (*Fleas*).—So far as is known, these do not greatly interest the veterinarian, unless they are parasitic upon him; but the rat flea, *Pulex cheopis*, transmits bubonic plague in man, and the human jigger flea, *Sarcopsylla penetrans*, digs its way in under the skin, particularly under the hard skin of the foot, and lays its eggs subcutaneously. The dog flea, *P. serraticeps*, is known to be the intermediate bearer of the cystic stage of the tapeworm (*T. cucumerina*). It is possible that fleas may transmit other diseases in animals. Other fleas are *Pulex irritans*, human flea; *P. felis*, the cat flea; and *Sarcopsylla gallinacea*, the fowl flea, commonly known as the "jigger," which attacks poultry about the head.

Flies.—Flies are greatly concerned, directly or indirectly, in causing diseases amongst domestic animals in South Africa.

The House Fly (*Musca domestica*).—The house fly, now scientifically designated *Musca domestica*, was formerly named *Musca vomitoria*, by reason of one of its dangerous and objectionable habits). When house flies are in swarms, feeding upon some filth or other, the food is sucked up hurriedly, to be afterwards vomited up when the fly has settled itself down comfortably somewhere, usually inside some building, to be sucked up again in a more enjoyable and leisurely manner. The place selected may be on food, and usually all that is vomited is not taken up, and what is left may contain more than sufficient organisms to cause disease if they are brought in contact with a susceptible animal. Careful investigations have been made, proving that such flies may, in addition to their vomit,

be the carriers of vast numbers of disease-producing organisms upon their bodies, especially upon their feet; and it has been shown that organisms can pass through the body of the fly without injury, and be present in their droppings in a form capable of producing disease. In all probability, house flies play a much greater part in the dissemination of animal diseases than is thought.

With the object of ascertaining how far flies are responsible for the transmission of swine fever, the house fly and other non-blood-sucking flies, as well as those which do suck blood, have been experimented with. House and lesser flies have been caged in transparent cones or muslin tubes and placed on the eyes of pigs suffering from swine fever, and having discharges attractive to the flies or which will adhere to their feet; then these flies have been ground up with sterile salt solutions and hypodermically injected into susceptible pigs. It has been proved that flies so treated contain the virus of swine fever for at least twenty-four hours. House flies have been fed (allowed to feed) on the blood of pigs suffering from swine fever, and then ground up and injected into pigs. The blood-fed flies were kept for varying periods of from one to seven days, and then the body of a single fly used, and in every case the fever was produced, showing house flies may be among the chief carriers of this disease. Further experiments proved that flies transferred from the eyes of sick pigs to those of healthy ones carried the infection into twenty-four out of thirty-two subjects to which they were applied.

The life history of the house fly is as follows: The mother fly lays from 120 to 150 eggs. These are deposited for preference into horse (stable) manure, but many other similar substances will serve the purpose. The eggs in less than twenty-five hours hatch out into maggots. These maggots or larvæ commence feeding at once upon the decaying matter in which they are hatched, and grow for four to seven days, and when they have completed their growth crawl away into some dry crack or crevice and turn into a chrysalis or pupa, from which in about another seven days a fly emerges. The entire life cycle only occupies a period of from fourteen to eighteen days; therefore, during one season a single fly may give rise to billions of descendants. The fly lives for about thirty days, and during this time may lay two or three batches of eggs, the first batch being laid when about ten days, the laying period extending from two to four days.

The most successful methods so far found out for destroying

flies are those aimed at it when it is in the egg and maggot stage, and when it is adult. Where animals are kept it is impossible in most cases to do away with all suitable breeding-places for flies, but this should be aimed at as far as possible. If manure can be carted away to the fields daily, it will assist greatly in keeping the flies in check. It has been found that 1 pound of borax dissolved in water will serve for destroying the eggs and maggots in 16 cubic feet of manure when sprinkled upon it. This is rather an expensive way. One that has been found most effective and that will keep flies down to a minimum, although extraordinary precautions are not taken with the breeding-places, is the following:

Dissolve 1 pound of arsenite of soda in 1 gallon of hot water. Dissolve 10 pounds of cheap sugar or 2 gallons of treacle in 9 gallons of water. Mix the two solutions together and stir well. Take some branches of trees with leaves that will not fall off when dry, dip these into the mixture, and hang near the ceilings (out of reach of animals) and other places where flies settle and congregate, and if these branches are renewed occasionally the flies about the premises will be hardly noticeable. The branches should be burned when they are finished with.

MYIASIS

Myiasis is a term denoting disease produced in animals or human beings through infestation with the larvæ of flies. Thus the skin maggot fly (*Cordylobia anthropophaga*) causes cutaneous myiasis, the horse bot fly (*Gastrophilus equi*) causes gastric myiasis, the nasal fly (*Œstrus ovis*) of the sheep causes nasal myiasis, etc.

Whilst many species of fly maggots are known to invade healthy tissues, a doubt exists in regard to others as to whether a wound or unhealthy condition of the part is a necessary preliminary to attack. Such doubt exists in regard to the maggots of various species of "bluebottle" and "greenbottle" flies in most parts of the world. These maggots are found amidst a mass of pus and festering matter, working their way gradually into the flesh of animals in various situations, especially around the anal parts of sheep, and they are commonly credited with ability to cause primary injury. However this may be, they certainly aggravate any previous unhealthy condition to an intense degree, and are therefore to be guarded against as far as possible.

With regard to the nature of the reproduction of many of the flies causing myiasis, whether they are oviparous (produce eggs) or viviparous (produce young), doubt exists with several of them. In certain cases it would appear to be almost safe to assume that they may do either one or the other, in the sense that in some instances the egg requires such a short time to hatch out. If a mother fly was unable for any reason to find a suitable host to deposit the egg upon, it might hatch in her own body, and when the host was found she would deposit a larva instead of an egg, as in the ordinary way.

WARBLES AND BOT FLIES

The family Œstridæ, to which the above flies belong, is one of the most remarkable groups of the natural order Diptera, both on account of the habits of the adult species and the peculiar life

histories through which the different genera pass before reaching maturity.

The family contains a number of genera which are parasitic upon mammals, from the elephant to the squirrel, and upon man himself. In the perfect state these flies do not feed, and the mouth parts are accordingly reduced. They live, when compared with other flies, only a short time, and death takes place soon after the completion of laying.

Warbles in Cattle.—In Europe this is perhaps one of the best-known examples of flies of this nature—in fact, it is impossible almost for anyone who has lived there, and who has been in any way connected with cattle, not to have seen at some time or other warbles in the hides of cattle, which are the larval stage of the fly known as the *Œstrus bovis*. So far as South Africa is concerned, this fly has never adapted itself to the country, although on very many occasions cattle have been imported from Europe into South Africa with maggots or warbles in their hides fully developed, some of which must have left their host in the ordinary way and fallen to the ground. As this condition in cattle is so well known, one cannot pass it by without reference, even though one's South African experience leads one to different conclusions, as to the manner by which the warble and similar larvæ of these flies gain entrance to their hosts' bodies and take up their predilection sites—to the conclusions that have been arrived at by many European authorities on this class of parasiticism.

Warbles in Man.—The skin maggot fly, as it is commonly called, exists more or less all over Africa, but is found in greater numbers in the central portion of the continent than it is at either end. In South Africa cases at various times have been recorded in the Transvaal and Natal, but I am not aware of any having been recorded in either the Free State or Cape Colony. In Rhodesia cases occur in some portions of the territory every season. Usually it is only an odd case or two that is heard of, but in other seasons, for no assignable reason, the cases are much more numerous and a fair percentage of the population are affected.

The fly that is responsible sometimes is called the Tumbu fly; it is a thick-set, compactly built fly about the size of, or slightly larger than, the ordinary house fly.

It is very lazy and easy to swat. It is brownish in colour, and is named *Cordylobia anthropophaga* (Grunberg). Grunberg suitably named it—*cordylobia* signifying "living in a boil," and *anthropophaga* "man-eating."

I am not aware if the life history of this fly has been fully worked out by any observer, but gleanings from the observations of victims, medical men, and others, I think, leave little more to be found out about it.

Whether this fly lays eggs or gives birth to a live maggot is unknown, but probably it is an egg that hatches out after being in contact with the skin for perhaps only a short time, and then it would appear that the maggot or larva hatched out bores its way into the skin directly over the spot where it came in contact with it. I think this boring in of the maggot must be painless or attended with very slight irritation, or one would expect so many of them would be killed before they could dig in that the species might be in risk of extermination. Experience and observation point to the habit of the fly depositing these larvæ, in the great majority of cases, in the sleeping-places and upon the wearing apparel of humans. Woollen under-garments hung upon the clothes-line to dry are a very favourite ground for the flies to deposit their eggs or larvæ upon. The number of warbles found upon those portions of the body covered by the undervest or by a cholera belt supports this theory, as well as the fact that maggots have been found on such garments. Sleeping-places are no doubt another favourite site for deposition; it may be on the bed-linen or pillow, or even on the planks of a native's bed. Broadly, I think it may be taken that anything that has been in intimate contact with the human body is regarded as a more or less suitable spot by the fly.

In addition to this somewhat roundabout way to get at a person, I think there can be little doubt that in some cases the flies deposit the larvæ direct upon the skin, and in support of this view the number of young babies that are affected on the scalp may be instanced; also in dogs and pigs. I think the method is frequently the direct one, and the site selected is in many cases in the scrotal region.

I have called this disease "warbles in man" for the simple reason that anyone who has seen warbles in cattle will recognise it at once.

Treatment.—The line of preventive treatment that is adopted is, in the first place, to swat all the brown and other sorts of flies that one sees, next keep the bed covered up by a mosquito-net or other cover, and it is claimed if all the under-garments are well ironed and baked in the oven after they have been dried, the larvæ are killed and infection does not take place.

In travelling in Central Africa, where this pest is prevalent, a

charcoal iron for ironing clothes after washing is regarded as an indispensable item of equipment.

After the larva has dug in it is said to remain stationary, but that only means that it does not shift from the spot. Judging from one's sensations, it is anything but stationary—in fact, it feels like an accomplished gymnast—but the intermittent pain caused by it is greatly due to where it is located. I had one under the arm that I thought must be located upon a nerve from the way it could twist me up. Others, again, cause little pain, but they all itch and irritate a lot. It is held that the warble remains in the skin for about fourteen days, at the end of which time it comes out the same way it went in, drops into the ground, crawls under some cover, and becomes a chrysalis, from which in due course the fly emerges and so completes the life history.

For the first few days the larvæ cause no irritation in the skin, and remain unobserved. With the commencement of the irritation the spot looks like a small blind boil, but if examined carefully a small hole will be found in the skin, through which the larva obtains air, and probably a small black speck, the fæcal matter of the larva, will be observed directly under this opening. Later on the opening will be very plainly visible to the naked eye, the black spot will be larger, and the movements of the grub will be visible. Treatment consists in pressing the grubs out, but this, the same as in the case of cattle, is difficult to do until such time as they are nearly ready to leave of their own accord, and whilst they are maturing their host suffers most aggravating intermittent pain. Personally, I prefer to risk making the place into a suppurating sore by digging them out or bursting them with a needle. Fortunately, the few that I was able to get at and treat that way healed quickly, and gave me no trouble.

Congo Floor Maggot Fly (Auchmeromyia luteola).

The early stages of the life cycle have been described by Mr. Fuller, who states:

"The eggs hatched in about fourteen days. With a view to testing their biting and penetrating power, I at once applied some of them to the skin of my arm. They almost immediately attacked the epidermis, rasping it away until the blood flowed. There was practically no sensation from the bite, although my attention was concentrated on the operation. The maggots did

not burrow, however, and as soon as they were engorged fell off, and buried themselves when placed in some sand. This process was repeated on several later occasions, but unfortunately I was suddenly called away from headquarters, and when I returned the creatures were dead."

As the name implies, the habitat of the fly is more central than South Africa, but the maggots have been found in Natal and Rhodesia, and very likely also in the Transvaal. The maggots, which are normally dirty white, become red in colour after a feed of blood. They are found in dirty native huts and hide during the day in crevices, etc., in the floor and about the walls, coming out in the night to suck the blood of the sleepers.

BOTS IN EQUINES

Bots, or *papjes* as they are called in Dutch, are the larval stage of species of flies called Gastrophilus, and are commonly found in the stomach of the horse, mule, and donkey.

Bedford, who has studied these flies at Pretoria, states: "Of the three species found, *Gastrophilus intestinalis* (*equi*) is the commonest, but both *G. pecorum* Fabr. and *G. nasalis* Linn. are far from being uncommon."

The fly in general colour and size greatly resembles the honey bee. The abdomen of the female is long and is bent forward underneath the body when the fly is at rest. The head is large, having a very broad face. The mouth parts, which are never used, are very rudimentary and inconspicuous. The eggs are deposited by the fly whilst she is on the wing.

The life history of the fly is briefly as follows: The female fly deposits her eggs on the hair, the favourite sites being the front legs, shoulders, and chest. These eggs are firmly attached to the hair by a glutinous material that encircles the hair, and are of a yellowish colour. In the course of a few days—seven to thirteen (Bedford)—the eggs hatch out into a larva or maggot, which commences to crawl about on the skin. Osborn, who studied the life history of the fly in America, came to the conclusion that the eggs did not hatch without the assistance of the horse's tongue. The theory held until recently was that this movement of the larva on the skin caused irritation to its host, thereby inducing it to bite the spot, by which means the larva obtained entrance to the mouth, and from there found its way to the stomach. The eggs or larvæ certainly do cause irritation, or, to be on the safe side, I will say that the animal can be seen to frequently bite those sites when eggs are present; but the latest theory is that the larva is independent, does not wait for the horse to give it a lift on the way to its destination, and that soon after it has hatched out of the egg it commences boring through the skin and makes a way to the stomach by another path than the gullet. This question of the route taken by the larva to its destination is a most interesting one. The most common site for

the bots to be found is in the stomach, but they are also occasionally met with at the back of the mouth, in the gullet, in the small bowel adjoining the stomach (duodenum), and in the back bowel (rectum). Without differentiating between the sites selected by the different species of bot flies to deposit their eggs, practical men are aware that a number of eggs are deposited on parts that the host cannot get at with its own tongue, and that are rarely, if ever, given a lick by a friend; therefore, considering the number of bots that are frequently found in an animal, it may not appear unreasonable to conclude that possibly the larvæ have more than one route to their destination. Bedford, in the report of his researches on *Gastrophilus nasalis*, states:

"The females lay their eggs on the hairs round the lower lip and those on the intermaxillary space between the mandibles, and have also been reported to have laid their eggs in the nostrils of their host.

"The ova, unlike those of *G. intestinalis*, do not require friction and moisture in order for them to hatch, as the young larvæ are able to break off the operculum without any assistance. Ova which were collected from horses and kept in tubes at room temperature hatched in a few days, whereas those of *G. intestinalis*, when kept under similar conditions, always failed to hatch, even after several months."

With regard to the bots that attach to the back bowel, there are usually *G. pecorum* or *G. hæmorrhoidalis*, and they are believed to attach there some time before being evacuated. Whichever way the larva goes, it gets to its destination, usually the stomach, and by means of two hooks anchors itself there for about ten months before letting go and passing out with the dung. On reaching the ground the bot wriggles into the earth under the dung or grass roots for cover, and its skin becomes hardened and forms a shining shell-case, from which a fly emerges in from thirty to forty days. Mating of the flies then takes place; the female flies deposit their eggs again on the hair, and the life cycle is completed.

From the foregoing it will be gathered that the life cycle takes approximately twelve months. It is well to remember this when judging the results of different medicines employed in the expulsion of bots. The season for depositing the eggs varies in different parts of South Africa. In the Cape it is said to commence as early as January, and in Rhodesia about July; and as the season lasts about two or three months, horses that are running in the veld may be said to be never free from bots. They are not

properly rid of the previous season's crop before the new tenants have taken possession.

This fact can be verified by making post-mortem examination of animals that have died from various causes at all seasons of the year. The damage to the host caused by bots is a matter of controversy. Some people maintain that bots eat through the stomach, and so cause the death of the horse. The late Dr. Hutcheon of the Cape was a great opponent of this theory, and went to considerable trouble to disprove it; and whilst admitting that after death a bot might wriggle its way into or even through the wall of the stomach, in life it did not do so. My experience leads me to believe that the doctor was right in his contention; at the same time, I do not agree with those men who contend that bots do no harm. In my opinion they are a frequent cause of indigestion and colic. There is one grave error that many people still fall into on opening a horse's stomach and finding bots present, and that is that the bots have eaten half of the lining membrane of that organ, whereas the stomach is quite normal. Half of the stomach is white and the remainder is of a pink colour, and to prevent making such a silly mistake people should take the trouble to acquaint themselves with the appearance of a normal stomach. With regard to the attachment of bots at the back of the mouth and entrance to the gullet and windpipe, Bedford states that in South Africa it is not an uncommon occurrence to find the larvæ of *G. pecorum* in this position, and quotes an extract from a report by Major Cannon, S.A.V.C., who carefully recorded the symptoms of a horse in his charge which was affected with larvæ in the pharynx, at the Base Veterinary Hospital, Luderitzbucht, South-West Africa:

"On arrival at my depot the horse stood with his head near the ground, and a profuse flow of ropy saliva was coming from the mouth, breathing slightly increased, temperature normal. Examination of the throat and submaxillary revealed no swelling, and no obstruction could be found along the course of the œsophagus.

"Upon elevating the head for the purpose of making an examination of the mouth, the horse struck out and stamped with both fore feet, walked backwards, coughed violently, and fell on his side, with glottic spasms, threatening to bring almost immediate death. He rallied, however, and in a few seconds got on his feet again, staggered about for a minute, and then came to a standstill with legs straddled, his head extended in line with the neck, facial expression distressful, much champing of the jaws, and rapid efforts at deglutition; clicking sounds were emitted,

and the salivary evacuations enormously increased. I came to the conclusion that the horse had some foreign substance lodged in his throat.

"After allowing twenty minutes' quietude the mouth was cleaned as much as possible of mucous accumulations, and by leaving the head in a position indicating most comfort to the horse examination was effected, without the presence of any foreign body being located. My interference, nevertheless, excited another fit of coughing, but with less alarming results. The mouth was flushed with a solution of boric acid and alum, a mustard liniment and cotton-wool pack applied around the throat, and belladonna and potassium chloride electuary prescribed for the tongue.

"Several hours later the horse was standing quiet, but listless and depressed. The salivary flow from the mouth maintained. Food was refused, but water eagerly accepted. All attempts to swallow caused the water to return through the nostrils, and efforts were made to restrain coughing. The following day I found his condition the same; any attempt to elevate the head was resented, and threatened to bring further paroxysms of coughing, so disturbance in this manner was carefully avoided, as it was noticed that the cough was less frequent and severe whilst the head was depressed. The temperature remained normal, no complications were presented, and no swelling of the throat became visible, although slight manipulation of this part stimulated deglutition and threatened to excite coughing.

"At the end of the third day no appreciable improvement in the animal's condition was indicated, so I decided to dress the throat with a dilute solution of tincture of iodine and glycerine. This was accomplished by means of a mop made of cotton-wool secured to the end of a stick. The applications caused the horse great distress, and upon withdrawing the mop I discovered numerous larvæ (some of these larvæ, which were forwarded to the laboratory for identification, proved to be *G. pecorum*) of the Œstrus family adhering thereto.

"A second and third application later brought about similar results. On the fifth day a marked improvement was noticed. Salivary evacuations were much reduced, cough less painful, the head was restored to normal position, but food was refused until the next day; then an attempt was made to swallow some gruel, a considerable portion of which was returned through the nostrils.

"The mopping process was continued with success, and gradually day by day the powers of deglutition were much improved and the appetite much restored.

"Necessarily, the horse had lost much in condition, but was ultimately fit for discharge in about thirty-four days from the date of admission to hospital. During this period I was favoured at intervals by the arrival of ten more horses, all presenting the same chain of symptoms already described, but in varying degree

of intensity. Having discovered the cause of their troubles to be the presence of Œstrus larvæ involving the pharynx, the application of the mop and iodine treatment was promptly pursued, and the disappearance of alarming symptoms became immediately progressive. In every case food, such as bran and grass, was refused for some days, but water was always accepted and preferred to gruels.

"In no instance was a discharge from the nostrils observed except when an attempt was made to swallow the liquids. Not one of the eleven cases brought under treatment registered a temperature of higher than 102·2°, and complications were not recorded. The cough in all cases was moist, and represented a series of expulsive efforts in rapid succession, the inspiratory efforts being, as it were, choked off."

Treatment.—The treatments to rid an animal of bots are many, and as a rule their effectiveness depends upon the season of the year at which they are employed. If they are used at the season when the bots are voluntarily releasing their hold on the stomach and passing out with the dung, the medicine may appear to be very efficacious, and this is the time that they are usually employed, for the reason that the presence of bots in the stomach is found out by observing one or two in the dung or adhering to the anus. If an attempt is made at other seasons to remove the bots the ordinary medicines employed fail to do so. The only method so far that is supported by results is the Italian one of administering 15 gramme doses of bisulphide of carbon in a gelatine capsule at intervals of two hours.

The treatment of bots indicated is of a preventive nature. Flies like the bright sunlight, and do not enter the stables to lay their eggs; but with horses stabled at night and allowed to graze by day they are also easily destroyed. Directly the eggs are observed upon the hairs they should be burnt by means of the ordinary singeing lamp, a wax taper or a candle or rag soaked in paraffin being rubbed over them.

SCREW WORM IN CATTLE

At the end of 1918 a myiasis in cattle appeared in the eastern portions of Southern Rhodesia, and maggots from the wounds set up were forwarded to Mr. Rupert W. Jack, F.E.S., entomologist to the Southern Rhodesian Administration. Mr. Jack in due course hatched flies from these maggots, and gave the following description of them:

"The fly itself is of the 'blue-bottle' type, the whole body being of a dark but brilliant metallic blue. The length of an average specimen from the front of the head to the tip of the abdomen is about 9 millimetres, or, roughly, $\frac{3}{8}$ inch. The sides of the face or cheeks are buff-coloured, and the wings are free from the dark markings shown by the common allied species (*Pycnosoma marginale*). Whilst the present species bears the characteristics of the genus Pycnosoma, its specific identity is unknown to the writer. The larva, or maggot, and the pupa are of the form shown in the plate, the greatest length amongst the maggots submitted being about 14 millimetres, whilst the average pupal length is about $11\frac{1}{2}$ millimetres."

The life history of the fly has not been worked out; probably it is similar to that of other flies of this nature. Mr. Jack states the pupal stage lasted fourteen days in the specimens submitted to him.

During the summer season 1918-1919 the disease was present to a greater or less extent in the eastern half of Southern Rhodesia, but no cases were recorded in the western portion of the territory.

Whether this disease only appears in cycles of years in certain places, or whether this must be regarded as a permanent spread of the disease, is unknown. The only other place in South Africa, so far as I am aware, where it has been reported as occurring is Swaziland. I am informed that it has certainly been present in that territory for at least six years, probably longer.

Nature.—Whether the fly deposits eggs or maggots on the animal is unknown, but from the number of maggots present in

one place it would appear as if eggs were in all likelihood the method of propagation. Likewise, whether it is essential that there shall be an open wound or whether a simple moisture on the surface of the skin proves a sufficient attraction for the fly and suitable medium for the larva to make its way through the comparatively unbroken skin into the muscles is undecided. Experience points to the latter as being sufficient.

The sites selected by the fly for attack may be on any part of the body where the skin has become broken or abraided. It is impossible to perform surgical operations such as castration or dehorning in the fly season without getting the wounds filled with maggots. Even branding is risky, as cases of infestation have occurred at the sites of the brands. In working oxen severe whip-marks have proved suitable sites. Ticks also appear to make conditions favourable for infestation, particularly the bont-legged ticks, whose habit it is to attack the animal in clusters, especially in the region of the udder and scrotum. Ticks in the ears of cattle also cause favourable conditions, and severe cases of infestation by maggots have been found in these regions.

The extent of the lesion caused by the maggots depends upon the time it has been in existence before being observed; a cavity as large as one's head may be found, but it is usually smaller, about the size of a teacup. Cases have occurred in bulls in which the testicles have been entirely eaten away and the scrotum filled with maggots. The cavity, when examined, is found full of dead tissue, pus, and maggots. After scraping this out and washing the cavity clean, if it is again examined maggots will be seen embedded in the flesh with only the tails showing and wriggling as the maggot " screws " its way into more solid flesh.

Treatment.—Cast the beast, and arrange that the wound is uppermost. With a blunt spoon scrape all the pus, maggots, etc., away; place a pipette (glass tube) in a bottle containing bisulphide of carbon until it contains about 15 to 20 drops of the solution. Place the forefinger over the upper end of the pipette and withdraw from bottle, then drop the solution into the wound, distributing same drop by drop by manipulating the forefinger at the top of the pipette. Let the beast remain down for some minutes to allow the bisulphide to vaporise, and as this vapour is heavier than air it will settle into all the cavities and kill the maggots with which it comes in contact.

Another method for this part of the treatment is, after scooping out, to wash the cavity out with petrol, which also kills the maggots. If neither bisulphide of carbon nor petrol is at hand,

the maggots may be removed from the flesh by a pair of sharp-pointed tweezers.

The next part of the treatment consists in endeavouring to prevent flies reinfesting the wound, and for this purpose Stockholm tar has been greatly used. Some people plug it with cotton-wool or tow, and dress all round and over the wool with Stockholm tar. Cheaper methods than the foregoing have been sought, and the following are favourably reported upon: Take coal-tar 1 quart, calcium chloride 200 grammes, and boracic acid 100 grammes; heat tar on a slow fire, and stir in the powdered ingredients. Use this as a dressing for the wound after it has been cleaned and freed of maggots. A still cheaper method is, after thoroughly cleansing the wound, to fill it full of wood ashes; this is said to help in the healing of it as well as preventing reinfestation by the flies.

SHEEP NASAL FLY (ŒSTRUS OVIS)

History.—The larvæ of this fly were first observed in the head cavities of sheep by Vallismieri in 1712. The fly became well known through the description of Bracy Clarck in 1797. The disease is found all over the world except in Australia, and is very prevalent in South Africa.

Nature.—The fly is not quite ½ inch in length; it is yellowish-grey in colour, and covered with a few hairs. The face is yellow; thorax brownish-grey, streaked with obscure lines; the abdomen spotted with white, yellow, and black, and covered at the tip with fine hairs; the wings are transparent, marked with three black spots at the base. Like the other Œstridæ, this fly lives in the perfect state during the warm weather, and it only flies when it is dry and warm. It hides in holes and crevices in the walls of sheep kraals, which it leaves when coupling time arrives. When fecundated the female fly goes in search of sheep, which are afraid of its approach, and the flock huddle together, circle round, and put their noses between their fore feet on the ground to avoid it. During the periods of cud-chewing the fly has a better opportunity of depositing its eggs or larvæ upon the nose. Authorities differ upon the point of whether it is an egg or a living larva. The probabilities are that it may be either one or the other. However this may be, the larvæ crawl from the nose up the nasal cavity into the maxillary and frontal sinuses, and even into those of the horn cores, where it attaches itself by means of the hooks upon its head and remains there for about ten months. This is the time that it has been proved to do so in other countries, and there is no reason to think that there is any difference in the period in South Africa. When the maggot is fully grown it loosens its hold and starts to crawl out of the nasal chamber, but it excites violent sneezing and snorting in its host, and is so expelled with a mucous discharge set up by the irritation caused by it. On reaching the ground the maggot buries itself in the ground, and in about twenty-four hours after leaving its host it enters into the pupal (chrysalis) stage, in which

it remains for from four to six weeks, when the fly emerges from the chrysalis in the usual way.

Goats are also affected.

Symptoms.—The symptoms will vary in intensity according to the number of maggots present in the nasal chambers, their situation therein, and the age of the animal affected. The most prominent symptom is the frequent sneezing and snorting, accompanied by a thick mucoid discharge from the nostrils; it may be from one nostril. It is most noticeable in the early morning in animals that are confined in a kraal or shed, as soon as they get up on their feet sneezing and the discharge of sticky mucus commences; the dried discharge will be seen around the nostrils, and frequently on the legs and shoulders. The irritation in the head causes the animal to rub its nose against its leg and other objects; occasionally the discharge may be streaked with blood.

The foregoing are the symptoms that are observed in the great majority of cases; in odd cases one meets with symptoms of a nervous nature. Observers in other countries have accused the maggots of occasionally entering the cranial cavity. Whether this is so or not I do not know; the cases that I have seen I have thought to be due to the temporary irritation of a nerve and of a transient nature. An animal may be seen to hold its head high and walk with high steps or in a circle. In other cases nervous excitement will be shown; the animal shakes its head, the eyes roll, and it looks as if it was going into a fit, but usually such symptoms pass away and end in a fit of sneezing.

The persistence of the symptoms depends upon the numbers and age of the parasites present. If there are only a few the animal will be all right in a week, but if there are a number of parasites of different ages the sneezing and discharge may persist for some weeks.

Goats are not so severely affected as sheep, although there may be a copious discharge from the nostrils; probably they are more clever in dodging the flies, or the latter may prefer sheep. Adult sheep as a rule are not seriously affected; they will lose condition during the time the maggots are leaving the head, and occasionally a few will die. The disease is more serious in lambs and younger animals; in these the discharge appears to be more copious, and frequently they appear to be unable to keep the nostrils clear by sneezing, and at times, particularly in a bad season and when they are in a weakly condition, a number die from broncho-pneumonia.

Treatment.—As a preventive treatment smearing the nose with Stockholm tar has been recommended; this is rather a tedious

operation in a large flock, and automatic application of it has been attempted by getting the sheep accustomed to take coarse salt from a long box or manger, and afterwards fixing a board in it with holes large enough to admit the noses of the animals to the salt cut in it, and when they have become accustomed to this by smearing the board with Stockholm tar.

Many kinds of treatment for the destruction of the maggot in the head have been tried, including snuff of various kinds, but with not very satisfactory results.

The treatment that has given the most satisfactory result, and that gives hope of being able to eradicate the disease from a herd, is corrosive sublimate, 1 in 1,000 of water. This is injected with a small syringe into each nostril, the sheep being held on its back for a few seconds to let the fluid get well up the passages. The injection must be repeated three or four times at intervals of a few days.

SHEEP BLOWFLY

This trouble is usually designated as "fly-blown sheep" or "fly-struck sheep." The latter appellation in South Africa is more commonly understood to mean animals infected with tsetse-fly disease—not necessarily sheep alone, but different classes of animals.

"Fly-blow" in sheep is caused by the larvæ (maggots) of two flies. The common "blue-bottle" (*Calliphora vomitoria* or *erythrocephala*) which invades the butcher's shop and the housewife's larder is responsible for some of the stricken sheep; the greater number are victims of the green-bottle (*Lucilia sericata*), the true sheep blowfly, which is smaller than the blue-bottle. The maggots of the blue-bottle are surface feeders, remaining on the skin, but the green-bottle's repulsive brood tunnel into the flesh and eat the sheep alive.

History.—The disease has been long known in Europe and Australia, but until the last few years it caused no serious trouble in South Africa. Prior to 1896 it was confined mainly to the humid districts of the east coast of Cape Colony and to a few centres in the Eastern Province. Since that date it has spread at times at rather an alarming rate, and now it is found during the damp warm weather in almost every part of the Union, but it has not yet been observed in Rhodesia.

Nature.—The fly deposits its eggs on the wool of the sheep, particularly on those parts where it is dirty, or where there are open sores or wounds; the crutch and in the neighbourhood of the tail are very common sites, especially in lambs that have suffered from diarrhœa and soiled the parts, but any part of the body can become infected. The eggs hatch out rapidly and behave according to their kind as described.

Symptoms.—The suffering animal appears restless, uneasy, shakes its tail in an excitable manner, and may try to bite the affected part. Upon examination a damp and fœtid patch of wool will usually indicate the site of the maggots.

Treatment.—Cut away the wool and clear out all the maggots. Be careful not to let any of these fall on the ground or escape;

put them with the wool in a tin or other vessel, afterwards burning the lot; then dress the affected parts with one or other of the numerous astringent preparations usually employed, or 5 per cent. carbolated oil. In South Africa the readiest applications to hand are usually sheep dips; these should be used in the proportions given in the directions for use.

Preventive Treatment.—Some thirty or forty years ago a trap for this class of fly was invented, that, when baited with strongly smelling meat, attracted the flies, which to get at the bait ascended an upward plane to enter the trap, and, once inside never discovered the aperture by which they entered. This method is used to some extent in Lancashire in England, and perhaps would be found helpful to a certain extent in South Africa; but here, the flocks being large and the conditions under which they are kept different, probably a better preventive treatment is to dip them once or twice during the season when the sheep are liable to be fly-blown in an arsenical sheep dip that will remain in the wool and on the skin for some time, and act not only as a repellent to the flies, but also destroy any maggots that may happen to be on the skin and in the wool.

Another line of treatment is what is known as "crutching." This consists in shearing the wool from the region of the crutch.

Probably a combination of all these measures, coupled with the burning or burial of all carcases that are found lying about the veld, would give the best preventive results.

SHEEP TICK, KED, OR LOUSE FLY (MELOPHAGUS OVINUS)

This is really a fly without wings. It is classed in the natural order Diptera, and belongs to the family Hippoboscidæ. It differs from the true tick in having only six legs, whereas the adult tick has eight. It differs also from the sheep louse, although its habit of living amongst the fibres of the fleece resembles that of the louse, whence the name "louse fly."

The ked never leaves its animal host, all stages being found upon it. The egg hatches within the body of the female, and is nourished there until full-grown, where it forms a roundish, white, or yellowish case, which is deposited by the fly, being fastened to the wool by a gluey substance. In this case the maggot changes to the pupa or chrysalis state, from which the fly subsequently comes out.

These larvæ or puparia are born one at a time, and only three or four are produced within the lifetime of the ked. The

greatest number known to be yielded by one ked is eight. The pupal stage lasts from three to twenty days. The ked lives for three or four months.

The ked is flat, with a bag-like abdomen; its length is about $\frac{1}{4}$ inch, often a little less. The head and thorax are square, or nearly so. The colour is brown, the abdomen being more of a greyish-brown. The ked is very bristly, being covered with hairs all over the body, particularly so on the abdomen and legs. The feet are furnished with strong hooked claws. They try to evade capture by running into the wool, and when caught cling tenaciously to it.

Thirty years ago keds were scarcely known in South Africa, but now they are very common in many of the sheep-rearing districts of the Union. They may be found on the sheep at all times of the year, but are particularly noticeable at shearing time. Their food consists of the grease (yolk) of the wool, the débris of the hair, and the blood that they cause to flow by their bites. Considerable irritation is caused by their presence; the yield of wool is less, and considerably stained, which reduces its market value. They cannot live many days apart from their host, except in the shorn fleece. At shearing time many of them leave the shorn sheep and take shelter upon the lambs, on which they prove veritable pests.

Treatment.—Dipping in arsenical dips twice at an interval of a month. The first dipping, if effectively done, should destroy all the adult parasites, and the second destroys all the parasites that have subsequently hatched out.

BLOOD-SUCKING FLIES

Blood-sucking flies have been accused of being the transmitters of disease in South Africa. So far as is at present known these flies, other than tsetse, do not act as an intermediary host to any parasite, and the transmission is only claimed to be of a mechanical nature; but even so, considering the number of diseases there are in this country in which the causal organism is in the blood circulation, the subject is one that should not be lost sight of.

The disease with which they have been to the greatest extent implicated is trypanosomiasis, but they have also been accused of transmitting anthrax.

Horse Flies.

Tabanidæ.—This family of flies, which is a large one, and includes the horse flies, blind flies, or gad flies, has had perhaps the greatest amount of accusation levelled against it.

The Tabanidæ more particularly frequent woods and pastures, and it is during the summer and the warmest hours of the day that they are most redoubtable. Their flight is rapid and accompanied by a buzzing sound. This noise usually alarms the threatened animal, but despite what it may do the fly settles upon it, and does not leave until it has engorged with blood. The bites of many species are very severe, often resulting in a runaway. After the fly has left its host a drop of blood frequently flows from the site where it inserted its proboscis, and a slight temporary swelling arises also. Tabanidæ lay eggs on leaves and other similar smooth surfaces, particularly in wet ground.

Stomoxys (*Stable or Stinging Flies*).—These flies are accused of transmitting anthrax; they lay eggs in heaps of heating grass or other vegetable matter, and in manure. The species that are accused of transmitting trypanosomiasis are *S. calcitrans* (the commonest species), *S. nigra*, and *S. hyperosia*.

Hippoboscidæ.—This is a small family, and only three species of it interest the veterinarian: one is the ked of sheep, which has

already been described; another is the *Hippobosca rufipes*, whose method of reproduction is similar to that of the ked, the only difference being that the larvæ of this fly do not hatch out on the host, but in the ground. This is the flat-bodied, reddish-brown and yellow fly that is frequently seen on horses, less often so on cattle, and runs about the skin like a crab, and owing to its leathery texture is very difficult to kill by the hand. Horses become accustomed to the presence of these flies upon their bodies, but even one of them upon a sensitive-skinned horse unaccustomed to these flies will nearly drive the animal frantic. They have wings and can fly, but they rarely leave their host, and live upon blood. The third species is *Hippobosca struthionis* or ostrich fly, which is very similar in appearance, life history, and habits to the *H. rufipes*. It is very troublesome to ostriches, and causes the birds to become restless and pick themselves, so that the feathers are often worthless.

Eradication.—The eradication of these classes of blood-sucking flies cannot be attempted on the same lines as those of the house fly by destroying their breeding-places and eggs, but experience has shown that a number of these flies disappear when the regular weekly dipping of cattle in arsenical dips is pursued. The habit of *H. rufipes* of refusing to be driven from its host results in its undoing when horses are dipped in the ordinary cattle-dipping tank and a few gallons of oil, which floats on the surface, has been added to the dipping solution.

TSETSE FLIES

THE name " tsetse " is a Zulu word, and is supposed to resemble the noise made by the fly when in motion. The flies belonging to this order, of which there are several, are known as Glossina. The species that are found south of the Zambesi are *Glossina morsitans*, *G. brevipalpis*, and *G. pallidipes*. The first named is the only one that has been recorded in Southern Rhodesia; the other two have been found in Portuguese East Africa; and the third is the one present in Zululand.

The word " tsetse " is not used for all the species of Glossina, which may be described as follows:

Ordinary-looking, sombre, brownish or greyish-brown flies, slightly larger than the common house fly; measured from the tip of the proboscis to the end of the closed wings they are $\frac{1}{2}$ to $\frac{3}{4}$ inch in length. They may be distinguished from all other blood-sucking flies by the combination of two characteristics—viz., (1) the proboscis is visible when the insect is viewed from above, projecting in front of the head. (2) When at rest the wings are closed the one over the other like the blades of a pair of scissors, not crossed like duck's wings as so frequently described. Several other species of blood-sucking flies have one or other of these characteristics, but none, except the tsetse flies, possesses both.

History.—The original description of *Glossina morsitans* West. dates as far back as 1850, and there is at least one traveller's reference to a fly whose bite was reported to be fatal to stock, nearly forty years previous to this (Austen). Gordon Cumming's notes on the insect in 1850 brought the name " tsetse " before the public. Livingstone, in his " Missionary Travels and Researches," in 1857 also described the effect of the tsetse's bite on cattle and horses. The work of Colonel (now Sir) David Bruce in Zululand in 1895, demonstrating the rôle that trypanosomes play in the diseases set up by such bites, stimulated interest in the subject, which was still further increased when, in 1903, Castellani in Uganda demonstrated that trypanosomes were present in the spinal fluid of negroes suffering from sleeping

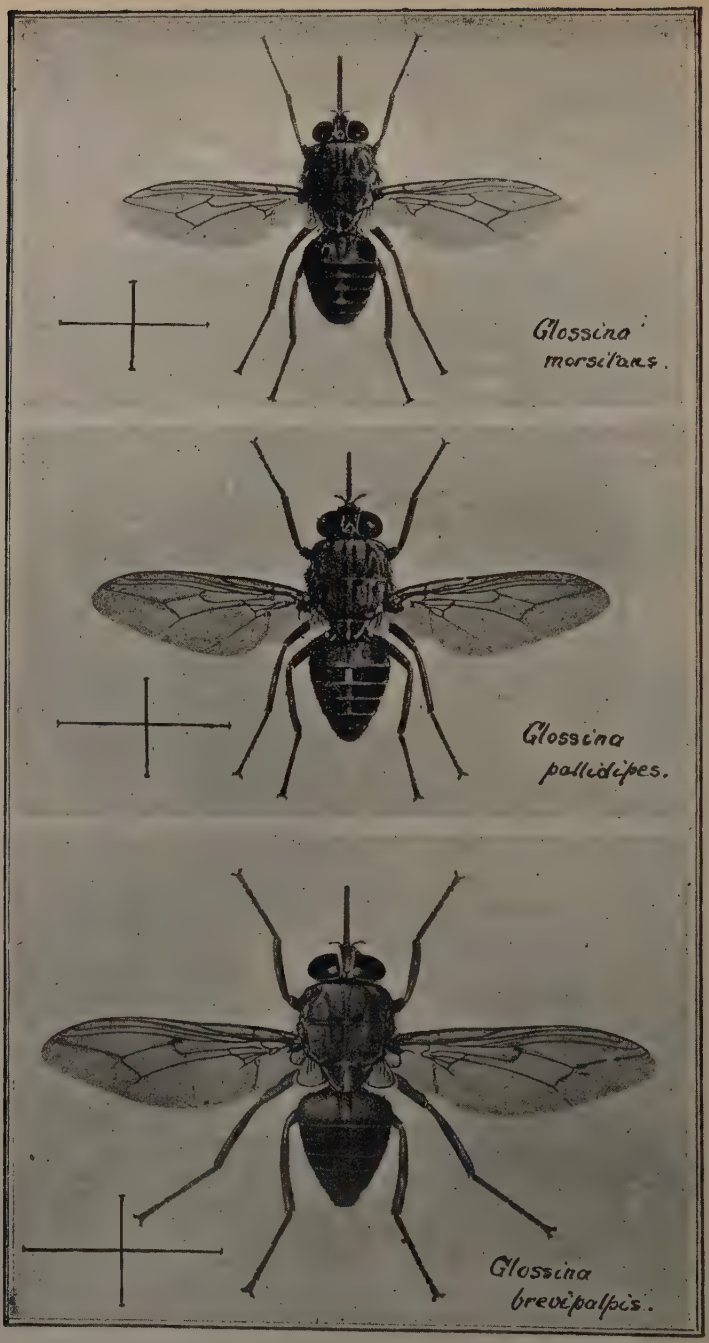
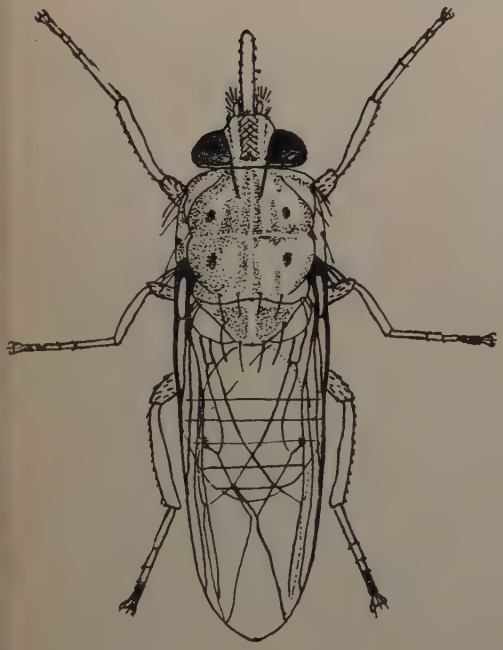


FIG. 20.—THREE SPECIES OF TSETSE FLY. (After Austen.)

The crossed lines against each figure indicate the natural size of the insect, the perpendicular line giving the length of the body (including the head), and the horizontal line the distance between the tips of the expanded wings.

1.

GLOSSINA MORSITANS
Enlarged 6 diameters
After Austen



2.

G LONGIPENNIS
Enlarged 4 diameters
After Austen

3.

G MORSITANS PUPA
Enlarged 6 diameters

4.

G. MORSITANS LARVA
Enlarged 6 diameters
Somewhat after Lloyd

FIG. 21.

1. *G. morsitans* IN A RESTING POSITION FROM THE SIDE. (Compiled from Austen.) The shaded portion shows the appearance of the abdomen in an unfed fly, and the dotted line indicates the distension produced by a full meal of blood.

2.—A SPECIES OF TSETSE FLY (*G. longipennis*) IN A RESTING POSITION FROM ABOVE, SHOWING THE POSITION OF THE WINGS AND THE PROBOSCIS PROJECTING IN FRONT OF THE HEAD. (After Austen.) The actual proboscis, or piercing organ, is enclosed in a sheath formed by the two palpi or feelers, and is in reality as slender as a fine hair.

3. PUPA OF *G. morsitans*, SHOWING THE PECULIAR PROCESSES AND NOTCH AT THE HIND END OF THE BODY.

4. LARVA OR MAGGOT OF *G. morsitans* AS EXTRUDED BY THE FEMALE FLY. (Somewhat after Lloyd.)

sickness, until at the present day an immense amount of knowledge has been obtained of the tsetse flies and the rôle they play in the transmission of human and animal diseases.

At the present time the only localities in South Africa (south of the Zambesi River) in which tsetse flies are found are Zululand, Portuguese East Africa, and the northern portions of Southern Rhodesia bordering the Zambesi River. Prior to the rinderpest wave which swept through a great extent of the subcontinent in 1896 to 1897, tsetse flies existed over a more

FIG. 22.—VEGETATION BORDERING MANZITUBA VLEI (AUGUST): TSETSE VERY ABUNDANT.

extended area. In the "Encyclopædia Britannica," 1888, appears the following statement: "A tsetse belt forty miles wide along the whole course of the Limpopo still bars the spread of European settlement beyond the Transvaal in the direction of the Zambesi." Our present knowledge of the habits of the fly, coupled with the statements of old hunters, would indicate that probably there were breaks in this belt that were free from the fly, but it certainly did exist to a great extent, particularly on the Macloutsi Shashi Tuli and Lundi Rivers.

Theiler, writing in the *Transvaal Agricultural Journal* in 1909, states:

"Nagana and the tsetse fly at the present time do not exist in the Transvaal. Up to the time of the rinderpest in 1897 the fly was known to occur in the low country; with the disappearance of the plague it also disappeared. The places frequented by the flies were principally along the rivers of the low country, more particularly those bordering the present game reserve. Some farmer friends of mine who were 'voortrekkers' have told

FIG. 23.—SHADY THORN TREES, SIPANI VLEI, SEBUNGWE (OCTOBER): TSETSE ABUNDANT.

me that not far from the Magaliesberg, north of Pretoria, some thirty or forty years ago flies were met with, and names like Vliegpoort would seem to indicate this, 'Vlieg' referring principally to tsetse. On one of the old maps of the Transvaal, issued by Jeppe, a line is drawn which separates the southern regions as free from flies from the northern as known to be fly infested. The southern point of that line was near the junction of the Marico and Crocodile Rivers, and it stretched in a north-easterly direction through the Waterberg to the Limpopo."

Simultaneous with the outbreak of rinderpest in 1896 to 1897, which destroyed immense numbers of game, especially buffalo, the tsetse disappeared from many of its old haunts, to the majority of which it has not returned. A belief is current that the imbibing of the diseased (rinderpest) blood caused the disappearance of the fly. This seems improbable, the more probable cause being the loss of the game, the natural food-supply of the insect. After the rinderpest was over it was found that tsetse, with the exception of one or two very small areas near the

Fig. 24.—Mopani in April: Tsetse present in Numbers.

Zambesi River, had disappeared from Southern Rhodesia. Since that time the game has increased considerably in numbers, and with this increase the fly has also extended its area, and as the extension is a continuous, if slow one, it is naturally attracting a considerable amount of attention.

Nature.—The tsetse is a particularly alert and active fly. It darts straight to its resting-place without any preliminary hovering. If its resting-place be the skin of an animal or human being and its object food, it rests for some moments, ready to

dart away again at the first sign of molestation. If satisfied that there is no immediate danger, it will commence to feed, often shifting its feet for a moment as if to settle them comfortably. The immediate effect of the insertion of the proboscis varies greatly, probably in accordance with the point striking a nerve or not. Jack states that he has watched a tsetse on his arm from the time it settled to the time it left engorged with blood, and was unable to detect its presence by sensation at all, even when he saw the beak inserted into the skin. At other times

FIG. 25.—"PAN" IN MOPANI (DECEMBER): TSETSE PRESENT IN NUMBERS.

the prick will cause the victim to start as sharply as if a hot needle had been inserted. An undisturbed fly gorges itself with blood until its abdomen, ordinarily bearing a shrivelled appearance, is distended in the form of a round bead. The insect possesses great suctional powers, being able to engorge itself in a very short space of time. After doing so it leaves its host and flies heavily to the grass or other suitable position near by.

Both the male and the female flies suck blood, which is contrary to the general rule with blood-sucking flies, and the female fly produces a single full-grown maggot at a birth, and not eggs,

as is customary with insects generally. Such maggots are produced only at the rate of about one a fortnight. This method of reproduction undoubtedly greatly influences the habits of the fly, their slow spread, and their occurrence in nature. The maggot at birth for a few hours is capable of movement, which it utilises to wriggle into a sheltered position and turns into a chrysalis, from which a fly emerges in the space of from one to two months. Each female gives birth to from eight to ten larvæ.

So far as investigation has gone only three things are essential

FIG. 26.—MOPANI IN OCTOBER; UNSUITED TO TSETSE AT THIS SEASON.

for the existence of the fly: they are blood, humidity, and shade. There is no reliable evidence that the tsetse flies ever feed in nature on anything except blood drawn from a living animal, and their habit of remaining near the watercourses and in moist vlei ground, particularly in the winter season, is thought to be due to the ever green foliage affording shade in such localities; in the summer season, when all the trees are in leaf, they are certainly found at greater distances from such localities. They also possess what may be termed a homing instinct; flies carried out of a fly-belt upon the bodies of persons or animals, when they leave their host fly back to the place where they were bred

over long distances. It has been shown by Dr. Lamborn in Nyasaland that they may return as great a distance as ten miles. The reason for the existence of this instinct is a matter of speculation; it may be due to the females when in a pregnant condition being unable to fly great distances, and that the majority of the flies carried out of the belts may be males. Whether this is so or not, its presence, coupled with the manner of reproduction, undoubtedly accounts for the flies being found in "belts" that are sharply defined in the winter season, in which they have been usually observed by hunters and others who have described them.

FIG. 27.—MOPANI GIVING WAY TO RIVER VEGETATION: APPROACHING A DANGER ZONE.

The flies feed mostly during the day-time; they may feed to a limited extent during the night, and more so on warm moonlight nights. This habit has been recognised for a number of years; therefore people compelled to cross a fly-belt with animals prefer to do so at night-time. They feed at all times during the day, but in very hot weather they are more active during the early morning before 8 a.m. and in the afternoon after 4 p.m. In the winter they are not very active until the sun has been up two or three hours. When the wind is high or cold they are not very active, nor during a shower of rain.

TRYPANOSOMES TRANSMITTED BY TSETSE FLIES

PROTOZOA are divided into five classes. One of these, the Mastigophora (or whip-carriers), is again divided into five classes, one of which includes the genus Trypanosoma (borer body). The disease caused by these parasites is known as trypanosomiasis.

The trypanosomes are pre-eminently blood parasites, and are common in all the great classes of vertebrates, but have not so far been found in invertebrates. A number of them are non-pathogenic.

History.—They were first observed in the blood of a trout in 1841 by Valentine of Berne. In 1843 Mayer found them in the blood of frogs. In 1845 they were described in the field-mouse and vole, and in 1850 in the black rat. They attracted hardly any attention until 1878, when Lewis discovered the *T. lewisi* in rats in Calcutta. The first pathogenic trypanosome was discovered by Evans in 1880 in India, in cases of the disease known as surra occurring in horses and camels. It was not until 1894 that the next pathogenic trypanosome was discovered by Bruce in Zululand, in the disease known as nagana, or tsetse-fly disease. In 1894 Rouget found a trypanosome in the blood of a horse suffering from "dourine" in Algeria. In 1901 Elmassian in Paraguay found a trypanosome in a horse with *mal de caderas* (disease of the hind-quarters). In the same year Dutton discovered a new trypanosome in the blood of a European resident in Gambia. In May, 1903, Castellani in Uganda discovered a trypanosome in the spinal fluid of negroes suffering from sleeping sickness, and since that time the knowledge of this class of disease has greatly advanced.

It is impossible at the present time to state the number of species of pathogenic trypanosomes known to science, because authorities do not agree on the point of what constitutes a specific difference.

Nature.—All the trypanosomes are actively motile, but the rapidity of movement varies in different species; they nourish themselves by osmosis solely, and never attack blood-corpuscles in a mechanical way.

The parasites can be changed in appearance, motility, disease-producing powers, and resistance to drugs, by passage through different vertebrate hosts; and in this lies, perhaps, the greatest difficulty in arriving at a classification acceptable to all authorities.

The common natural method of infection is by the bite of an insect. Bruce, in 1914, classified the African trypanosomes pathogenic to man and domestic animals into three groups, comprising nine species. His classification in its entirety is accepted by few students of the subject, but whilst the classification of the trypanosomes is in such a state of flux Bruce's arrangement, for the reason that it is perhaps one of the most practical ones, may as well be taken. Besides taking into consideration the appearance and structure of the parasite, and their disease-producing powers, he also considers their development in their intermediary host, the tsetse fly, and arranges them as follows:

A. The *T. brucei* group. This comprises the polymorphic parasites. They develop in the gut of the fly, and then pass to the salivary gland, where they complete their development.

B. The *T. pecorum* group. Small and monomorphic. Development takes place in the intestines and proboscis, but the salivary glands are not invaded.

C. The *T. vivax* group. Monomorphic and very active. Development takes place in the labial cavity and trypophanynx, but not in the intestine or salivary glands.

"Dourine," which is a disease due to a trypanosome, was discovered in 1894 in Algeria, and latterly in the Union of South Africa; its intermediary host or bearer is not a fly. This classification is apparently incomplete, but it serves a useful purpose in showing the relation of tsetse flies to the majority of diseases in Africa due to trypanosomes. The relationship of tsetse flies to the spread of trypanosomiasis is a most important one, but a matter equally so from an economic point of view and more alarming is the question of the possibility of the transmission of the disease by flies other than tsetse. South African experience supports the presumption that without tsetse the transmission of the disease from one animal to another does not take place. Innumerable instances can be quoted where animals affected with fly disease have been allowed to mix freely with healthy animals without any spread of the disease taking place, but latterly in Southern Rhodesia and in South Central Africa outbreaks of disease have occurred in places where tsetse flies have not been found; and although in many cases tsetse flies have

been present within a radius of some few miles, in others they have only been known to exist very many miles away, making it apparently a practical impossibility for them to have transmitted

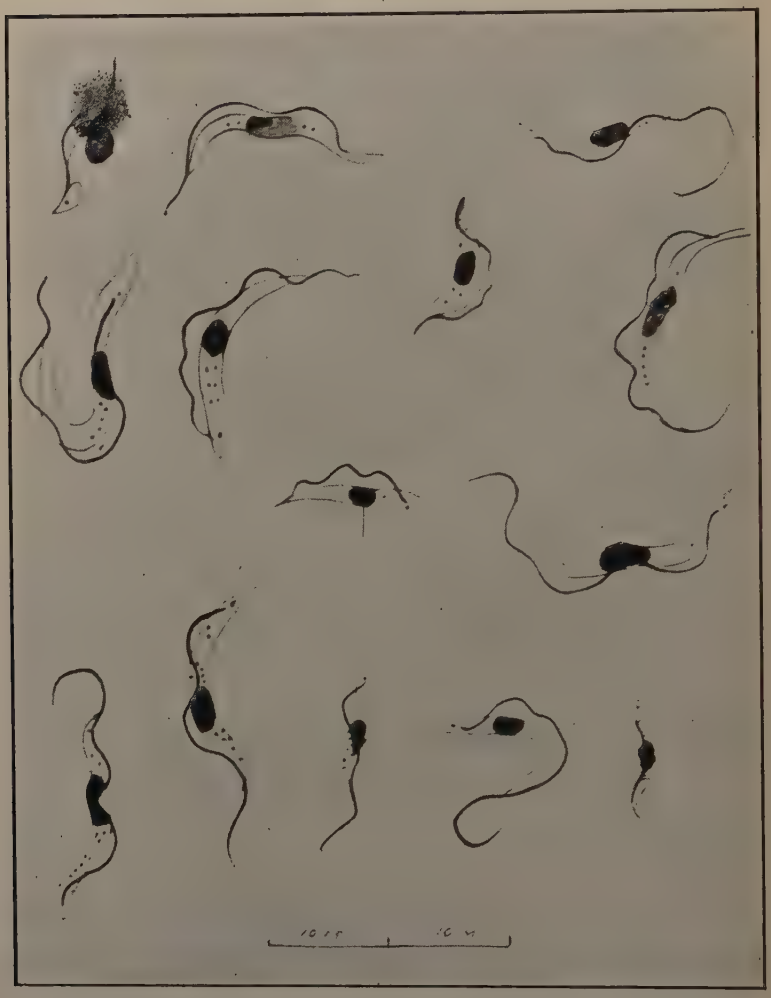


FIG. 28.—*Trypanosoma brucei var. rhodesiense.* (Drawing by Bevan.)

the disease; therefore there seems to be some grounds for supposing that the disease may be conveyed by other biting flies in a mechanical manner. Considering that the disease can be

easily injected by the hypodermic syringe, there seems to be nothing improbable in this contention, but it would appear to depend upon the existence of certain conditions. The most important would appear to be the species of organism, and whether the organisms are present in the blood in large or small numbers. This would depend greatly upon whether the case was an acute or a chronic one. If it was the former and the blood was teeming with organisms, and given a number of biting flies present, first biting an infected beast and then a healthy one

FIG. 29.—MULE SUFFERING FROM TRYPANOSOMIASIS. (Photograph by Bevan.)

only a few feet away, the probability of mechanical transmission seems to be great, and the possibility of a trypanosome being accidentally carried by the proboscis or saliva of a biting fly is even greater. On the other hand, in chronic cases the organisms are very few, and it is frequently impossible to demonstrate their presence in the blood by microscopical examination. In such cases mechanical transmission of the disease would be rare, even if it ever occurred. It would also appear possible that the motility of the trypanosome might govern the possibility of mechanical transmission to a considerable extent; for instance,

the *T. evansi* and the *T. brucei* are very much alike in size and structure, but the former shows greater movement under the microscope, and is transmitted ordinarily by mechanical transmission. *T. brucei* is, so far as is known, never so transmitted.

South African experience is largely built upon observation on travelling cattle and other animals that have passed through fly-belts, and it is possible that by the time they reached their destination and were mixed with other animals they were in a locality in which biting flies capable of mechanically trans-

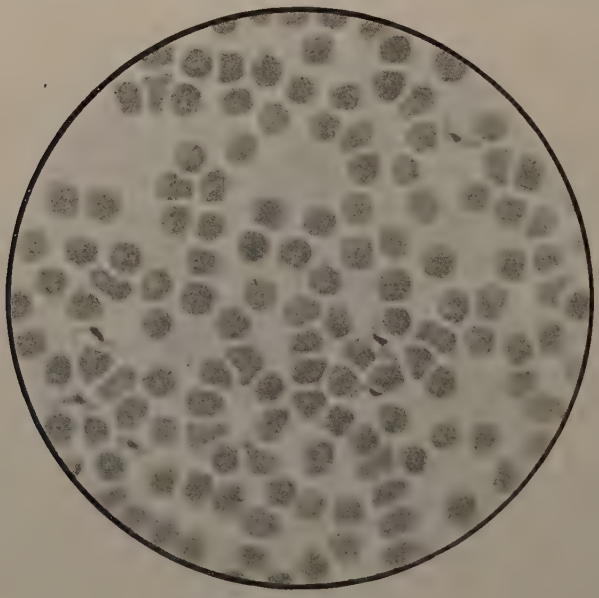


FIG. 30.—MICROPHOTOGRAPH OF BLOOD CONTAINING TRYPANOSOMES. (Photograph by Bevan.)

mitting the disease did not exist at all or in any appreciable numbers. There may be some other explanation, but no doubt one day the difference, which is to a certain extent a contentious subject, will be satisfactorily cleared up.

Hosts.—So far as South and Central Africa are concerned, and with the exception of dourine, the wild game of the country are the principal hosts or carriers of the trypanosomes, and to very much less extent the domestic animals may act in a similar manner. Amongst the game the "big game" are the principal hosts, but it is probable that in fly-belts all the herbivora and

many of the carnivora act as reservoirs of the organism. It has by inoculation experiments been demonstrated as existing in several different species. But not all ruminants in the fly-belts are susceptible or carriers, and the same remarks apply to tsetse flies; it has been held that only about 50 per cent. of the former and 5 per cent. of the latter do so. Further, it is believed that certain species of wild animals are commonly the reservoirs for different species of trypanosomes, but that the various tsetse flies are capable of acting as intermediary hosts for all the trypanosomes; therefore it follows, and has been observed, that when different orders of the domestic animals are sent through a fly-belt, distinct infections are set up. On the other hand, the reverse may have occurred, and cattle only have contracted a fatal trypanosomiasis, while horses, mules, and donkeys have escaped, although bitten by the fly. Other cases have occurred in fly areas where the cattle have died out and sheep, goats, or dogs have lived.

It has been proved that after tsetse flies have fed on an infected animal they are capable of transmitting the disease to a healthy animal by mechanical infection, by means of the saliva injected when biting, for a period up to about forty-eight hours, and that it is not until twenty or thirty days have elapsed that they are able to transmit the disease by the trypanosomes that have undergone development within their own bodies.

Animals Susceptible.—Man and all the domestic mammals are susceptible to one or more of the different trypanosomes, but it is claimed that if an animal is infected through the bites of tsetse with two species of trypanosomes, both of which are pathogenic for it, the one less so will disappear from its blood in the course of a few days, leaving the more pathogenic one in absolute command.

The names of, and opinions regarding, the different trypanosomes frequently change as more is found out about them, but the three following are at the present time regarded as the common ones found in domestic mammals in South and South Central Africa:

T. brucei is very fatal to equines, smaller ruminants, and dogs, but is almost non-pathogenic to cattle.

T. pecorum is the commonest cause of trypanosomiasis in cattle, but it is also pathogenic for the other domestic mammals.

T. vivax resembles *T. pecorum* in the forms of diseases it causes in stock. Dogs are generally immune to its ill effects.

To these may be added *T. rhodesiense* (the cause of human

trypanosomiasis of Northern Rhodesia), which was first studied and described by Bevan.

Treatment.—One gramme of tartar emetic in a 4 per cent. aqueous solution for adult cattle and horses, injected intravenously every fifth day, has given good curative results with certain strains of *T. vivax* and *pecorum*, but no drug has been found to have any curative effect on *T. brucei* in equines.

Another line of treatment that has been found successful is one intravenous injection of 1 gramme of tartar emetic, followed by a course of nine powders of arsenious acid: three of 1 gramme, then three of 1·5 grammes, and then three of 2 grammes, administration being on alternate days.

Symptoms.—Described by Bruce in horses suffering from nagana in Zululand:

"The first symptoms of a horse being affected by nagana are that his coat stares, and there is a watery discharge from the eyes and nose. Shortly afterwards a slight swelling under the belly or puffiness of the sheath may be noticed, and the animal falls off in condition. The hind extremities also tend to become swollen, and the various swellings fluctuate, one day being considerable, another day less marked, or having disappeared. During this time the animal is becoming more and more emaciated. He looks dull and hangs his head; his coat still stares, becomes harsh and thin in places; the mucous membranes of the eyes and gums are pale, and probably a slight milkiness of the cornea of the eyes is observable. In several cases and in the last stages the horse presents a miserable appearance. He is a mere scarecrow, covered with harsh rough hair, which has fallen off in places. His hind extremities and sheath may be more or less swollen, sometimes to a great extent, and he may become quite blind. At last he falls down, unable to rise, his breathing becomes shallower, and he dies exhausted. During his illness he has shown no signs of pain, and up to the last day has had a fairly good appetite."

Of cattle he says:

"The general symptoms in cattle are less marked than in horses or dogs. They gradually waste away; the hair, at first harsh and staring, tends to fall off; there is some trickling of a watery fluid from the eyes and nose, and a tendency to diarrhœa. In many cases the dewlap becomes swollen and baggy."

There is sometimes ulceration of the cornea and corneal opacity.

"*Post-mortem.*—Very few well-marked changes are met with on post-mortem examination. Enlargement of the spleen is perhaps

the most marked lesion. There is generally some gelatinous infiltration under the skin and mucous membranes, and between the muscles, and a quantity of fluid in the heart sac, and in the lung and abdominal cavities."

Montgomery and Kinghorn in Northern Rhodesia gave the following clinical picture of cattle suffering from the disease:

"At some period there is the appearance of emaciation and dulness, the coat harsh and hidebound, head drooping, eyes dull and watery, but petechiæ on the conjunctival membranes were rarely noted, and occasionally there is a nasal discharge."

Bevan gives the following symptoms of the more typical cases met with in Southern Rhodesia in cattle:

"The animals have a hard, dry coat, the hair standing on end, and appearing very coarse and dusty. In places the skin falls off in flakes. The animal is hide-bound, 'tucked up,' and shows no inclination to lick itself. Tears run from the eyes, and sometimes strings of mucus from the nose and saliva from the mouth. In nearly all far-advanced cases there has been noticed great weakness of the hind-quarters, the animal swaying and dragging its legs. It is often too weak to lift its feet to avoid a stone. The muscles of the hind-quarters waste away, the anus is retracted, and the sphincter seems to lose its power, so that fæces accumulate in the hollow beneath the tail. Chronic diarrhœa is frequently met with, together with the loss of power to regulate the discharge of the fæces, the animal's hind-quarters becoming foul and scalded. The animal feeds greedily. Even in those cases where it is unable to stand up it will lie, feeding the whole time, even up to a few hours before death. The mucous membranes are anæmic, and the heart weak, fluttering, and extremely fast."

From the foregoing it will be observed that, although the principal symptoms are similar, there are differences between the pictures of each of these observers. It must be remembered that they were working many hundred miles apart, and local conditions or disease caused by different trypanosomes or even different strains of the same organism must be allowed for. The symptoms expected to be found in the typical "fly-struck" animal are emaciation, weakness, harsh coat, with falling off of hair or skin, pale mucous membranes, accelerated pulse, and thin watery blood. But there is another type of the disease which has also been observed by Bevan, and that is in which animals in good condition die in the early stages when the temperature is in the vicinity of 106° F. Such deaths have recently occurred in an outbreak of the disease on the Gwaai River in Matabeleland.

The temperature is usually described as a fluctuating one, the rises being coincident with the presence of parasites in the blood. On this point Bevan states: "Experience in this country (Southern Rhodesia) seems to indicate that there is little, if any, relation between the presence of parasites in the peripheral blood or glands and the temperature or clinical condition of the animal. Chronic and lingering cases seldom show an elevated temperature, and not infrequently a subnormal one, even in the extreme heat of the day." The symptoms in the sheep, goat, dog, and pig are similar to those seen in cattle and horses.

Period of Incubation.—The period of incubation appears to be about ten days, more or less, probably depending to a great extent on the virulence of the particular organism causing the disease and the natural resistance of the animal affected. As has been seen, animals may succumb to the disease in its early stages with the first rise in temperature, before they have lost condition, but it is usual for them to linger on for weeks or months. Animals that contract the disease during the summer season frequently do not die until the first rains of the following spring. In this connection it may be pointed out that wetting the body—whether it be by rain, crossing a river, or by other means—of a fly-struck beast is mostly attended with fatal consequences.

Eradication.—The question of the eradication of tsetse-fly-transmitted diseases is almost sure to engage attention within the next few years, if for no other reason than making it possible to open up to civilisation large stretches of good country that are at present debarred by reason of the existence of what are known as tsetse-fly diseases; and in connection therewith it must be remembered that no matter what has been the experience of other parts of the world, the South African experience is, the existence of tsetse flies and pathogenic trypanosomes are so intimately associated that extermination of the former brings about disappearance of the latter. This disappearance has been well exemplified when rinderpest destroyed a great proportion of the game and tsetse fly, and trypanosomiasis disappeared with the game. The game as they exist in nature, in herds of hundreds and at times thousands, disappear with the advances of civilisation, and become reduced to herds of much smaller dimensions; and it is doubtful if any other conditions outside of actual game reserves will ever obtain, but that is quite a different matter to the indiscriminate slaughter of game for the purpose of ridding land of tsetse-fly disease. The only other known

method of dealing with tsetse fly is the destruction of shade, but this is too expensive a process to be practically applied; besides, few, if any, would like to see these two known processes applied in any other way than by the gradual advance of civilisation. But these methods are slow and entail losses to those who can ill afford them; therefore if it is desired to open up the natural resources of South Central Africa it behoves all, and particularly those who take an interest in the preservation of the fauna of Africa, to bring scientific brains to bear on the subject. When it is remembered how difficult the tick problem appeared and how it has been overcome, the tsetse-fly problem may not be so stupendous if it is tackled in the right manner, and it is possible that dipping might solve the latter, as it has the former.

SURRA.

Synonyms.—Surra is the name which was given to this disease by the natives in that district in India in which it was first discovered, and means "rotten," "dried up," etc. In other provinces it has other names, but conveying a similar meaning. In the Soudan it is called "mbori."

History.—The causal trypanosome was discovered by Griffith Evans in 1880, and has been named after him the *T. evansi*.

The disease is extremely widespread in India, and exists in Burma, Manipur, Assam, Persia, and French Indo-China. It appeared in the Dutch East Indies in Java in 1900, in horses and buffaloes, and has now become enzootic there. In 1901 the disease was reported in the Philippines. It exists in North-East Africa and in other places in the world. The disease was introduced into Mauritius through slaughter cattle imported from India in 1902, and from July to October of that year 1,882 solipeds and 1,681 cattle are reported to have died there. So tremendous were the ravages which it caused in the island that almost the entire equine population was wiped out, and the cattle were reduced to very small numbers. In 1902 some camels were imported into the Transvaal from Somaliland. On arrival they were found to be suffering from some unknown disease. Theiler, by injecting the blood of these animals into dogs, was able to set up trypanosomiasis, and found that nearly every one of the camels was a carrier of this parasite, which he believed to be the *T. evansi* and the disease to be surra; therefore he had all the animals promptly killed and buried. His diagnosis was afterwards confirmed by Laveran of Paris, the renowned authority on trypanosomes. Theiler also carried out a series of experiments under proper precautions with various species of animals, including horses and cattle, and found that the cause of the disease in every way corresponded with that of mbori. Further corroboratory evidence was also forthcoming in that the camels were bought at the conclusion of the war against the Mad Mullah, and it was known that some 20,000 camels had been imported for war purposes from India, which on the termination of hostilities were

disposed of. Surra is not dependent upon tsetse flies for its spread, and but for Theiler's foresight and prompt action South Africa might have suffered a similar experience to that of Mauritius.

Nature.—The causal organism, *T. evansi*, closely resembles the *T. brucei*—in fact, so much so that it may be impossible to distinguish between them by microscopical examination. In bacteriology it is accepted that immunity is specific—that is to say, immunity which protects against the subsequent inoculation of a given bacterium shows that this particular bacterium is identical to the one which was the cause of the immunity. For example, if an animal has recovered from quarter evil, say, and it is afterwards injected with quarter-evil virus and resists the disease, it is accepted that the organism which caused the disease and the one injected are one and the same. If this principle is accepted in protozoology it is proved that nagana and surra are separate and distinct diseases, and there appears no doubt that they are so. At the same time it should be remembered that in horse-sickness an animal may be immune to one strain of virus, but that another strain may prove fatal to it.

The disease is transmitted by biting flies other than tsetse. "Tabanus" and "hæmatopota" are the principal disseminators of the disease, but even "stomoxys" are capable of carrying the trypanosome from a diseased to a healthy animal by acting as simple vectors. Fleas can transmit surra from dog to dog, from rat to dog, and form rat to rat. Dogs frequently contract the disease by eating the carcases of animals that have died from it.

The question whether the fly invariably transmits the disease in a mechanical manner or whether in some instances the parasite may not go through some form of developmental cycle in a fly is a much debated one. The evidence, so far as it goes, appears to show that almost invariably the disease is transmitted in a mechanical manner, but it is possible that flies may act occasionally as real intermediary hosts. In the absence of experiments a decided opinion cannot be given on the point. Most mammals are susceptible to inoculation, and so far as can be at present ascertained all the more important ones may become naturally infected. In India, where the disease is enzootic, none of the mammals appear to escape infection. Horses, mules, donkeys, oxen, buffaloes, camels, elephants, goats, sheep, dogs, pigs, and deer may be attacked, and rabbits, rats, guinea-pigs, monkeys, and mice of all kinds can be readily artificially infected.

The mortality from the disease varies greatly in the different

animals. In the horse it is invariably fatal; a similar result occurs in donkeys and mules. Cattle living in countries where the disease is enzootic and widespread are endowed with great tolerance of the parasite, and generally show little evidence of its presence in the system. Indian cattle and buffaloes are of this description; the parasite is commonly present amongst them, and naturally they act as great reservoirs of the disease.

It is also a serious disease in camels, but amongst the other animals mentioned it follows a course similar to that in cattle. At times it is severe amongst dogs, particularly European bred ones.

Surra occurs exactly in those situations which are most favourable to the breeding of, and suitable as a habitat for, biting flies.

The surra belts are low-lying, marshy lands, subject to inundation and subsequent partial drying up, and covered with rough grass and brush jungle, and it is such areas that are best adapted to the Tabanidæ.

Surra appears in such belts immediately after the break of the rains, and continues during the rainy season. It is at this time of the year that the flies breed out, and are present in great numbers. It is only during the time that, and so long as, the biting flies remain prevalent that surra continues to spread amongst horses and camels.

It is a matter of common knowledge that these animals can live in such zones at other times of the year when no biting flies are present.

The parasites are present in the blood in great or small numbers in much the same way as in South African trypanosomiasis, and different strains of the parasite have been noted, or perhaps it would be more correct to say that the virulence of the organism is increased or decreased by passage through certain animals; thus the transference of the disease from a horse or a bullock to a horse results in an acute form of the disease, but from a camel to a horse the disease assumes a more chronic form.

Symptoms.—The symptoms in surra are the same as those described as occurring in nagana; the paralysis in the hind-quarters and anus, as described by Bevan, is present. The only difference in the symptoms described is that in the horse nettle-rash is occasionally noticed on the skin, and blood-markings are present on the mucous membranes, particularly on the haw of the eye.

Incubation.—The period of incubation is generally regarded as averaging about five days, but in some cases it may be as long as a month.

Treatment.—So far as treatment is concerned, the only preventive one that has yielded anything approaching success is the injection of human serum, and needless to say the method is impracticable. The curative treatment is in about the same stage as that for nagana.

Eradication.—In countries in which the disease is enzootic the problem of eradication, owing to the carriers—principally buffaloes, cattle, and camel, and the various flies capable of transmitting the disease—would appear to present greater difficulties than the eradication of nagana from South Africa; but our experience in Rhodesia leads to the conclusion that the regular weekly dipping of animals tends to greatly reduce, and in some cases entirely eradicate, certain species of biting flies. Perhaps relief may be obtained by the adoption of such a procedure.

DOURINE

Synonyms.—Common Venereal Diseases of the Horse, Horse Syphilis, Genital Glanders, Breeding Paralysis, Covering Disease (French, *Maladie* or *Mal du coit, Exanthème coital;* Dutch, *Slapziekte*).

History.—The disease appears to have been known in Algeria since early history; it was first described by Ammon, based on his experience (1796 to 1799) in the Prussian stud at Trakehenen. It is recognised in Northern Africa, including Egypt, in the whole of Spain, and Southern France, Austria, Hungary, and Italy. It is at present the only known pathogenic trypanosome indigenous in Europe. The early home of the disease would appear to be Syria and Asia Minor. Persia is affected, and probably parts of Arabia. The disease has been in existence no doubt for many years in India, but it was only in 1903 that Pearse demonstrated the actual cause.

A multitude of terms have been used to designate this disease. The names given were those that appeared most descriptive to the various observers. It was not until 1894 that Rouget found in Constantine, in the blood of a stallion affected with dourine (*i.e.*, dirt, unclean copulation), a trypanosome in which he immediately suspected the cause of the disease.

The disease was first recorded in Illinois in the United States of America in 1899, and was said to have been introduced by the importation of a French stallion. In 1903 it was encountered in Illinois and Nebraska, and also in Chile. The principal focus of infection, however, existed on the Indian reservation of South Dakota, where, on examination of the horses, 511 were found to be infected, 277 suspected, and 1,889 stallions had to be castrated. The energetic measures adopted appear to have been crowned with good results, as since 1906 no other cases have been noted. The disease appeared again in 1911 in Iowa, and in 1912 in Montana. Since then its existence has been determined in ten States, in which, during the period 1913 to 1915, a total of 109,159 horses were tested by the complement-fixation test, of which 4,699 gave positive reactions. From the States the disease

was conveyed into Canada, where stringent measures by the Veterinary Department have stamped it out.

In 1918 the disease was diagnosed in the Union of South Africa in Griqualand West, where its introduction and spread has been attributed to an imported Percheron stallion; but as the disease is now known to have existed in South-West Africa during the German régime, and still exists there, infection may have been brought in from that territory. The disease undoubtedly appeared in Griqualand West prior to the war, although it was not definitely diagnosed before 1918.

Nature.—Dourine is a progressive wasting disease complicated with nervous degeneration, manifested primarily in the genital organs, and due to direct infection by the *Trypanosoma equiperdum*. Natural infection appears to be entirely confined to breeding equines. The disease is most common amongst horses, but donkeys acquire it in a less virulent form. Dogs are susceptible, but cattle are immune to infection. Rabbits, rats, mice, and European dogs are easily susceptible to experimental inoculation, and may subsequently carry on the disease by coitus.

The *T. equiperdum* strikingly resembles the *T. brucei* (nagana). Like the other trypanosomes, it is a blood parasite, but it also causes local lesions, in which it remains for a more or less prolonged period. It differs from them in that contagion is conveyed by direct inoculation from the diseased to the healthy animal during coitus, and as far as is known at present no species of biting insect is concerned in its transmission; but it has been experimentally proved that biting flies can transmit the disease, so such a method of infection must not be entirely ruled out, but as the organism as a rule only exists in the blood in small numbers, the risk of the spread of the disease by this method under natural conditions is not great.

Artificial transmission of the disease is easily effected by smearing infected material upon a mucous or even a raw surface, so similar transmission by ordinary flies is a matter of possibility. The disease has been seen in geldings, presumably transmitted by contaminated sponges or the hands of attendants.

The parasite is found in the seminal and vaginal fluids of infected animals, also in the testicles, preputial glands, sheath, and in external ulcerations of the genital organs. It is, as a rule, difficult to find in the peripheral circulation, but it may be found in the " plaques." It is capable of living in the vaginal mucus for a considerable time without the mare showing any symptoms,

consequently such animals are dangerous and fruitful sources of spreading the disease.

Symptoms.—The disease generally justifies its old name of syphilis, as the symptoms and methods of infection are very similar; furthermore science, by the discovery of the causal organism of syphilis, has brought these two diseases even nearer together. The symptoms may be divided into three very distinct stages, termed primary, secondary, and tertiary.

Primary.—The sexual symptoms are the first to appear, and in mares the first indication that anything is wrong is the appearance of a discharge from the vagina, which wets the tail and hocks, usually so much that it attracts attention, but it may be so slight as to escape notice. It is such cases that frequently spread the disease. The external genitals are sometimes swollen, the mucous membrane of the genital passage is reddened, and nodules and ulcers may make their appearance upon it, while there is a tendency to frequent urination, which starts whenever the mare is made to move. During this stage of the disease the affected mare may appear to be in season. In stallions the first symptom may be a discharge from the urethra, accompanied by a swelling of the sheath; the animal will get frequent erections, and the sexual desire is increased. Swelling of the penis and scrotum follows, the swelling extending forwards towards the front legs. Swollen and extended nodules may form on the mucous membrane of the penis, followed by round, flat ulcers, which later on may heal, leaving white spots behind. The testicles are tender, and the legs, particularly the hind ones, are swollen. There is a tendency for nettlerash to occur; there may be a weakness of the loins, with a tendency to arch the back; but there are no symptoms of constitutional disturbance, the temperature being only very slightly raised, and the appetite remains good.

Secondary.—Some four to six weeks after the initial onset the general conditions will become more acute. The swellings are more noticeable and harder on pressure, emaciation is evident, and the animal becomes listless and tires easily. About the fortieth day the "plaques" or dollar spots first appear in the skin; they are characteristic, round or oval in shape, from 1 to 4 inches in diameter, not hot or painful, and have been described as if a disc of metal had been slipped under the skin. They may appear on any part of the body, but more frequently on the sides of the chest and croup; they last for an hour or two up to two days, when they disappear, leaving no trace behind them. The

loins exhibit pain on pressure, the animal seems slack in the hind-quarters, straddles the legs a little, and occasionally stumbles with one hind foot, and stops in its walk before going on again. This condition may go on for weeks, but in some cases the slackness of the hind-quarters rapidly increases, sometimes alternating with spells of apparent improvement, while sometimes one hind leg seems to be more seriously affected than the other, and is moved as in a horse suffering from stringhalt. Lassitude is more marked, and there is a difficulty in rising. It is difficult to induce a trot, and in the stallion erection is feeble and incomplete. The discharge in mares becomes viscid and irritating, and excoriates the skin of the thighs.

Tertiary.—From about the tenth month of the disease all symptoms become more acute. The appetite is variable, the emaciation is very marked, the animal gets worse and seems to lose control more and more of the hind legs, which are carried forward in walking with difficulty and evident pain; the horse may even sway whilst standing still. These conditions become aggravated, the animal lies down, has great difficulty in rising, and finally it falls and fails to get up again.

Treatment.—A similar treatment of arsenical preparations and tartar emetic as applied in nagana has been claimed to effect cures in a certain percentage of cases, but the malady is one that calls for prevention and eradication more than for curative measures. The disease is highly contagious, and although animals affected with it may apparently become convalescent and seem to recover, they are subject to relapses, and their continued presence amongst healthy animals may lead to the general dissemination of the disease throughout the stud. It is claimed that in animals that have undergone a course of the foregoing treatment relapses rarely occur, but the admission that they do occur is sufficient to condemn the practice for a great majority of outbreaks.

Very early castration is said to arrest the disease if the operation is performed while the affection is entirely local. This, coupled with an agglutination test, isolation of suspects, and destruction of affected animals, would apparently be South Africa's method for dealing with the disease.

OTHER TRYPANOSOMES

Amongst the other pathogenic trypanosomes, brief mention may be made of the following:

Mal de Caderas.--This is a disease affecting horses in sub-equatorial South America. It is caused by the *T. equinum* (*T. elmassiani*), which greatly resembles *T. brucei* (nagana), and is characterised by loss of power in the hind parts as the most striking symptom, similar to what occurs in dourine. The disease cannot be reproduced by feeding susceptible animals on fresh blood. The natural mode of infection is unknown.

Mal de Zousfana and El Debab.—This is a disease affecting horses and camels in Northern Africa. It is caused by *T. soudanense*, which is indistinguishable under the microscope from *T. evansi* (surra). The symptoms are those common to trypanosomiasis. It is transmitted by Tabanidæ.

T. congolense (Broden, 1904).—This is found in the Congo Free State, causing disease in cattle, sheep, and goats. It is transmitted by *G. palpalis* and *G. morsitans*.

T. dimorphon (Dutton and Todd, 1904).—Found in Gambia and Northern Rhodesia; it affects horses, cattle, sheep, and goats, and is transmitted by tsetse flies.

Sesheke Sickness.—A disease that made its appearance between Livingstone and Sesheke in Northern Rhodesia in 1908, and was said by Owen, who investigated it, to have been caused by a trypanosome of the dimorphon type, and to have been transmitted by Tabanidæ. It affected cattle only, and not horses and dogs, and only occurred at the season of the year, February to May, when Tabanidæ were plentiful. When Owen took up the study of " Sesheke sickness " in 1912 the annual number of cases must have totalled 1,000. The method of dealing with the outbreak was to segregate all the obviously infected cattle, and by the summer of 1914 no further cases occurred.

Soumaya.—This is a disease of horses, also affecting cattle and sheep in the French Soudan. It is caused by the *T. cazalboui*. It is transmitted by tsetse flies.

Baleri.—Caused by the *T. pecaudi;* it is usually a fatal disease of horses, occurring in certain localities of the Soudan.

T. nanum.—Produces a disease in cattle, sheep, and goats in the Soudan. The method of its transmission is unknown.

Sleeping Sickness in Man.—This is caused by the *T. gambiense* (Dutton, 1902) and the *T. rhodesiense* (Bevan, 1909), both of

which are transmitted by tsetse flies. It is believed that game is the reservoir of the disease.

Leishmaniasis.—Leishmaniasis in the dog is an important disease, because it is considered to be the reservoir for infantile kala-azar in young children. It is due to protozoal parasites termed " Leishman Donovan " bodies. At one time they were classed with the trypanosome, but are now classed as leishmania.

The leishmaniases are widely distributed in Asia and the countries bordering the Mediterranean Sea.

The disease is believed to be transmitted from the dog to the child by fleas. In the child and the dog it centres in the spleen and the bone-marrow, and death generally occurs.

COCCIDIOSES

THIS is an important group of diseases occurring in the domestic animals, especially in the bird and rabbit, and probably more rarely in cattle, sheep, goats, dogs, and cats.

Like the trypanosomes they are protozoa, and unlike them they are not blood but cell parasites, usually attacking the epithelial cells, especially of the intestine, bile-ducts, and urinary bladder.

They have been studied for the last seventy or eighty years, but, as in the case of the trypanosomes, there is still much to be found out about them and disputed points to be settled. Their life history, if complex, is very interesting, as it includes both asexual and sexual development; and as knowledge of it makes the diseases they cause more easily understood, I shall reproduce the notes that I took when attending Sir John McFadyean's post-graduate course in London some few years back.

Life History of Coccidia in General.

Of the whole of the known Coccidia this is briefly as follows: From the body of an infected host oocysts escape by different ways; these represent fertilised female individuals, which are intended to produce a specially resistant form of the parasite capable of withstanding the injurious conditions encountered in the outer world, and by so doing to ensure the maintenance of the species and to provide for the spread of the infection to new hosts.

The oocysts are provided with a resistant envelope, inside of which are spores; also with a highly resistant envelope, and within this again are a variable number of sporozoites. When these (sporozoites) obtain a fresh host they penetrate into the epithelial cells, and each starts to grow. This stage has been called the trophic stage, and the parasite is called a trophozoite; and when full size is obtained it proceeds to multiply by asexual division known as schizogony, and the parasite is known as a schizont. This division results in the formation of a large number of club-shaped motile bodies (resembling the sporozoites)

called merozoites. By this dissolution of the schizont the merozoites are set free, and each seeks out a new epithelial cell, into which it bores. It grows and again becomes a schizont; this is called the multiplicative division of parasites (it corresponds to division by fission in bacteria), which serves to overrun epithelial cells in the host, and therefore makes the disease serious. This asexual multiplication is not maintained indefinitely; it gives way to propagative multiplication when this is about to happen. Various merozoites, instead of becoming schizonts, become sexually differentiated, thus producing special coccidi called microgametocytes, which fall into a large number of motile elements called microgametes (which play the part of spermatozoa, the male element). Other merozoites develop into large coccidi called macrogametes, which assume an ovoid and a resistant envelope. Either before or after this envelope is formed each one becomes fertilised by a single microgamete, and this, then, is the oocyst (started with) intended to be extruded from the animal body.

Animals Affected.

Coccidia have been found in man and in practically all the domesticated mammals and birds, also in several wild ones. In England it is one of the commonest diseases. Found in tame rabbits, it is commonly present in the disease known as "pot-belly"; it also exists in these animals in South Africa, and at times has caused considerable losses in rabbits required for laboratory purposes. But so far as South Africa is concerned, I think a consideration of the diseases caused by these parasites in poultry and cattle will suffice.

COCCIDIOSIS IN THE FOWL

Synonyms.—Protozoal Enteritis, White or Chalky Diarrhœa of Chicks, Spotted Liver, Blackhead, Dysentery, etc. In South Africa it is often called South African Fowl Sickness.

History.—Coccidia in birds was first described by Rivolta in Italy in 1878. It has been found in practically all species of birds in the United Kingdom—in the fowl, turkey, goose, pheasant, grouse, crow, blackbird, etc. The disease is rare as affecting fowls in England, but is an exceedingly common disease in artificially reared pheasants. It is widely spread in Europe, Asia, America, Australia, and is very prevalent in Rhodesia and South Africa, generally amongst fowls.

Nature.—Coccidiosis in the fowl is an enteritis or inflammation of the lining membrane of the intestines. It may be acute, as is generally the case in chicks, or chronic in the chicks that survive an acute attack, and in older birds. In certain outbreaks many of the birds are found dead before the disease has been noticed; in other cases they are ill a few days and then die. In adult fowls, however, in the more chronic stages of the malady they may linger for a few weeks.

The causal parasite is blunt, oval in shape, and is known as *Coccidium* or *Eimeria avium* or the *Coccidium rivoltæ*.

Here again, as in the case of trypanosomes, doubt exists as to the identity of the parasite, whether coccidiosis in the fowl is always due to a distinct species of coccidia, or whether the rabbit parasite is transmissible to fowls. The experiments to prove this point have had contradictory results.

Chickens a few days old are very susceptible, and the mortality is then very great, especially in those hatched in incubators. The disease is more prevalent in the spring season, in the hot dry weather, and is usually contracted by the bird picking up the parasite from the ground with its food or drinking water.

Methods of Spreading.—The fruitful source of the disease is the purchase of birds, apparently healthy, giving off the parasites from their intestines and contaminating the poultry yard, pen, or aviary. Old cages, baskets, crates, fowl-houses, incubators or rearers, and such like, purchased at sales, sometimes introduce the disease. Eggs soiled with fæcal matter containing the parasite and eggs containing the parasite in their interior are sources of spreading the disease, so are " day old " chicks. It is supposed the coccidia gain the interior of the eggs before it reaches the terminal or shell-forming portion of the oviduct. They are found in the albumen, and in a hard-boiled egg can be seen with the naked eye as small, greyish-brown, blackish, or yellowish specks reaching to the diameter of a pin's head. Pigeons and wild birds coming from another place may introduce the disease either by harbouring the parasite within their own bodies or by their feet having become contaminated with the parasite at the place where they last fed, and shedding it when they again settle.

Symptoms.—In chicks only a few days old the appetite is suppressed, they are restless, the comb or beak turns a leaden hue, and they quickly succumb. In chickens a few weeks old they appear dull, droop their heads, and the disease is markedly manifested by a whitish or chalky material (white diarrhœa) adhering to or soiling the cloaca. When the disease is slight the

fæces are soft and brownish-yellow; in very acute cases yellowish and very fœtid.

In chronic cases the featherless parts are pale, which just before death assumes a bluish tint. Paralysis of both legs often occurs. The bird stands in a very listless, sleepy manner, and the mouth often gives off an offensive odour; it gradually gets weaker until it succumbs. Some cases pass into the chronic state, others die suddenly. The principal symptoms of the disease may be stated broadly to be diarrhœa and emaciation.

Post-mortem Appearances.—The carcase is usually emaciated, and around the vent will be soiled with fæces. The lesions are usually confined to the intestines and liver. In acute cases of the disease the intestinal lesions are not very striking to the naked eye; sometimes there is congestion, at other times there is none. When lesions are present they are more frequently confined to the large intestines, particularly to the cæcum (blind gut), but the small intestines are also not uncommonly affected. The changes are inflammatory ones; the lining of the cæca may have been shed, and the organ, one or both, may be considerably distended and contain an offensive yellow pasty material. In chronic cases the gut wall is from seven to ten times its normal thickness. In very young birds the liver is usually very much enlarged; in older birds it is frequently enlarged and studded with spots, yellowish or white in colour, about the size of a kaffir-corn seed or pea. These may be, and undoubtedly are, often mistaken for tubercular nodules.

Treatment.—Mr. H. H. Curson, M.R.C.V.S., who has dealt with the disease in the Cape, recommends the following:

Curative treatment is, in the acute stages, unsatisfactory. In chronic cases the ideal treatment would be the administration of some of the coal-tar derivatives. Naphthalin or its derivatives, alpha- and beta-naphthol, on account of their insolubility, are the most promising, as these are parasiticides, antiseptics, and sedatives, and also check the diarrhœa which so exhausts and debilitates the bird. The dose varies from 1 to 3 grains, according to the size of the bird and frequency of administration. The powder may be given as a pill, and along with either castor or olive oil ($\frac{1}{2}$ to 1 teaspoonful).

A tonic and astringent, such as iron sulphate, can be given in the form of Douglas mixture. This is prepared by dissolving 1 ounce of iron sulphate in a quart of water, then add 1 dram of diluted sulphuric acid, and of this mixture put 2 teaspoonfuls in a quart of drinking water, which must be kept in a shady spot.

Another remedy that has given considerable satisfaction is the addition of powdered catechu to the drinking water in the proportion of 15 grains of catechu to a gallon of water; continue for fourteen days, and at the end of that time very few or no parasites at all should be present in the fæces.

It must be remembered that the birds are extremely anæmic and in a weakened state, therefore diet must be liberal and nourishing; finely chopped or minced meat, boiled linseed, etc., should not be omitted.

Preventive Treatment.—In dealing with this disease it is important to remember that the excreta of diseased birds contains the parasites. To eradicate this complaint, the following directions should be strictly adhered to:

1. All dead birds to be buried deeply or destroyed by burning.

2. It is known that coccidia live for twelve months, possibly much longer, after having been evacuated by an infected bird. It is therefore difficult to disinfect a poultry-run, and a change to fresh ground is necessary. If, however, the infected stock is moved to fresh ground, that ground will rapidly become infected. The old stock should, therefore, be left where it is, any birds which show symptoms of the disease being promptly killed. Only newly hatched chicks should be placed on the new ground, and all brooders, coops, feeding and watering utensils should be thoroughly disinfected before use. There must be no communication with the old infected yard. In this connection it must be remembered that a person may unknowingly convey infective material to the new "run" on his boots.

3. If hens are used for hatching, there is a danger that they may infect the chicks; but if hens are preferred, then broody hens must be secured from a clean brood.

4. If these suggestions cannot be carried out, or if fresh ground cannot be provided, the best procedure is:

(*a*) To kill all birds exhibiting the first symptoms of the disease.

(*b*) To take off about 3 inches of the top soil of the most frequented parts of the runs, and disinfect or burn it.

(*c*) To disinfect thoroughly all houses, roosts, laying houses, food and water utensils.

(*d*) To use only healthy hens for setting.

(*e*) To place eggs in a solution of 90 per cent. alcohol, or first wash in methylated spirits, allow to dry, and then dip in perchloride of mercury (1 to 2,000) before setting them.

If the disease is non-existent, then all new arrivals should be viewed with suspicion and kept isolated until it is certain they

are free from the disease; microscopical examination of the fæces is the only reliable method of ascertaining.

Newly purchased eggs for setting purposes should be first washed in methylated spirits, allowed to dry, and then dipped in perchloride of mercury (1 to 2,000).

The incubator trays, drying boxes, brooders, market baskets, crates, cages, and any articles likely to convey infection should be thoroughly cleaned and then disinfected.

On farms, and wherever possible, a fresh site for coops and hatching boxes, etc., should be selected every year, and the old ground treated as recommended above.

Method of Disinfection.—It will be sufficient to wash the incubator trays and drying boxes with a 5 per cent. solution of lysol, hycól, or other carbolic preparation, and then allow to dry.

Coops, hatching boxes, brooders, perches, cages, and fowl-houses should be scrubbed with boiling water and caustic soda. When dry, the woodwork, etc., can be limewashed, and to the wash add some disinfectant, such as hycol.

The floor should be scraped and the scrapings burnt. Then may follow either the sprinkling of freshly slaked lime or spraying with disinfectant. A very serviceable spraying fluid is paraffin emulsion, which is easily prepared thus:

Hard soap	½ pound.
Boiling water	1 gallon.
Paraffin	2 gallons

First dissolve soap in the water, and then add the paraffin and stir vigorously; finally, dilute with four times the amount of water. The advantage of spraying is that the liquid more easily penetrates the crevices and corners. A good plan is to paint the perches and uprights and crevices with boiling tar, as ordinary lime wash is soon rubbed off.

As poultry farming is becoming more important every day, and owners are realising the value of well-bred stock, the first lesson that should be learnt is cleanliness. Any time spent on cleaning the fowl-run is well worth the labour, and at the same time disease and financial loss are reduced to a minimum.

COCCIDIOSIS IN THE OX

Synonyms.—Protozoal Gastro-enteritis, Bloody Flux, Red Diarrhœa, Red Dysentery, Hæmorrhagic Enteritis.

History.—Although dysentery or bloody flux in cattle has been recognised from antiquity, it was not until 1877 that Proger, a

veterinarian of the Berne district (Switzerland), reported observations made upon calves which died in two or three weeks with symptoms and lesions of acute enteritis. Pieces of intestine were sent to Zurn, who found a parasite which he regarded as a gregarine. Afterwards Railliet and Lucet, Zschokke, and others considered it a coccidium, which, on account of its specificity for cattle, was called the *Coccidium bovis*. To-day it is also known by the name of *Eimeria zurni*.

Bovine intestinal coccidiosis is especially diffused in Switzerland. It has been reported in France and in Germany. In Italy cases of it have often been seen in Piedmont, in Emilia, and in Venetia. In 1898 Gair observed it in Scotland. In 1905 Ducloux observed it in Tunis. In 1910 Montgomery encountered it in calves and badly nourished young stock in British East Africa, and in the same year Jowett discovered it in a calf at Rosebank, Cape Town. In Australia it was observed by Clelan, and in North America by Schultz and Th. Smith. In 1919 it was discovered in Ireland by Dolan. It is impossible to say to what extent this disease exists in South Africa. Bloody dysentery is of common enough occurrence; therefore it seems probable that the disease is more frequently present than is imagined; at the same time, it would be unwise to assume that all cases of red dysentery are due to the presence of coccidia in the bowels.

Nature.—The prevailing type of the parasite in the ox is said to differ from the ovoid forms found in the rabbit, but some investigators have claimed to have transmitted the coccidia of the ox to the rabbit; others, again, and perhaps they are in the majority, claim that the parasites are separate and distinct. The disease is mostly seen in animals between six months and two years old; it is said to be particularly grave in animals between ten and eighteen months. Smith has seen it even in sucking calves. It presents enzootic characters in young animals and sporadic ones in old. It seems to prevail mostly during the warm season; in Europe from July to September, and to disappear about October. As could be expected, it appears more prevalent on particular pastures, where the disease recurs from year to year, and it has been seen in animals during the housing period of the year.

The disease is taken into the system by means of food or water. All veterinarians are agreed in recognising that marshy waters and grass of humid meadows are particularly dangerous. The mortality varies in different localities in Europe; it usually averages between 5 and 15 per cent., but may go up to 25 per

cent. In Montgomery's observations 10 to 20 per cent. succumbed. The disease is almost always grave or mortal in young animals; the mortality is greatest in calves, older cattle being more resistant, and adults much more so. The better the condition of health, the more resistant to the disease. The disease in its acute form may be mistaken for rinderpest.

Symptoms.—The affection begins with diarrhœa, the fæces being discharged without much effort on the part of the animal, or at other times it begins with the emission of blood-clots without any other symptom. At first there does not appear to be much general disturbance of health, loss of appetite, or elevation of temperature, but later on the diarrhœa changes to dysentery, there is frequent straining, with passage of small quantities of fæces, composed almost entirely of grey-coloured shreds of mucus and clots of blood, the blood continually becoming more abundant. There is fever and irregularity or cessation of rumination. After each defæcation there is an effort of straining, the animal standing with its back arched and the tail sticking out; it may exhibit colic and frequently grind the teeth; the temperature rises to 105° or 106° F.; it visibly becomes emaciated, the flanks fall in, and the eyes sink in the head; it shows weakness of the hind-quarters and a notable difficulty in rising.

In some cases the course of the disease is extremely rapid, and death may occur in four or five days; in calves in even less time than that. In mild cases in which the appetite is not lost recovery may take place within a week. In cases in which the acute symptoms have continued for ten days or more the animal may assume the recumbent position, with a subnormal temperature, imperceptible pulse, and death supervenes in such cases: on the other hand, if recovery takes place its course is usually slow.

Post-mortem Appearances.—There is generally emaciation of the carcase, pallidity of the muscles, and soiling of the skin with fæcal matter. At first the lesions are limited to the rectum, the mucous membrane of which is thickened, hyperæmic, and sprinkled all over with blood-spots. Later, on the fifth or sixth day of the illness, the cæcum and colon are also involved; and in cases in which the animal dies after eight or ten days from the initiation of the process all the intestines appear involved. The large bowels usually contain very little fæcal matter; the mucous membrane is reddish-brown in colour and swollen, often lying in longitudinal folds and covered with mucus. At times this mucus is found in patches, of a somewhat resistant nature,

attached to the mucous membrane, but from which it is easily detached.

Treatment.—As a curative treatment Smith advises the following mixture: Powder of acacia catechu 8 grammes, powdered asafœtida 4 grammes, tincture of opium 8 grammes, creosote 4 grammes, and water 850 grammes. This makes one dose, which is given in gruel and repeated twice daily.

San Lorenzo recommends thymol in doses of 10 to 15 grammes. It is claimed this treatment has given cures in four or five days.

In addition to the foregoing treatment, the nursing of the patient would considerably influence the result. The animal should be placed in a stable and made as comfortable as possible. A dose of castor oil for a start is indicated, and apparently has been found to do good by Dolan in Ireland. Hay and other bulky foods should be withheld, and only nutritious and easily digested foods should be allowed; and gruel (flour or oatmeal), milk, eggs, and linseed tea should be given to the animal in a bucket if it will take it, and if not it should be drenched with it, and Smith's prescription given above should be mixed with it twice daily.

The preventive treatment lies in good sanitation and disposal of manure and other discharges of affected animals in such a way as to prevent spreading the disease to other cattle, and keeping young stock from grazing on known infected pastures.

SECTION III

DISEASES CAUSED BY WORMS

OTHER countries can no doubt be found that suffer more from worm infestation of man and the domestic animals than South Africa does, but it must be admitted that we have a full share of these pests.

The causes that are favourable for such infestation are many; the prevention and treatment are dealt with under the particular disease. Here it is only proposed to deal with the matter on broad lines.

The Cestodes, or tapeworms, are particularly prevalent in South Africa, and when a knowledge is acquired of the nature and life history of these parasites, coupled with the habits of the people and the methods of farming pursued, the reason for this being so is patent to all.

Broadly, the life history of tapeworms may be said to be that the larval forms of these worms inhabit the bodies of herbivorous animals, and the adult forms the bodies of carnivora. The latter, by eating the former, become infested with the parasites; and the herbivora, by eating or drinking material that has been contaminated by the bodies, or excrements therefrom, of the carnivora, become infested with the eggs and larval forms of the worms.

Man becomes infected with tapeworms by eating flesh in an uncooked or underdone condition, and in races that are in the habit of eating meat in this state the disease is particularly prevalent. The eating of meat that has only been smoked or dried may be mentioned as a fruitful source of infection. With regard to the eating of insufficiently cooked meat, and, in fact, the eating of all meat, whether it has been killed or simply found dead, the native races are great culprits, and consequently they have to pay the penalty of being frequently infested with tapeworm.

Native cookery at the best of times lends itself to the propagation of verminous infection. When the natives have a plethora of meat, such as a dead ox, the habit is to make a fire near the

carcase and to keep on throwing bits of meat upon the hot coals; these get burnt, perhaps to a cinder, on the outside, but the inside is quite uncooked, and any worms that may be present there in the cystic stage are entirely unharmed. One method that is practised by the Matabele has always appeared particularly revolting to me, and that is the cooking of a hare. The animal is simply put in the pot, hair and all, and boiled. The hair comes off with the boiling, and is skimmed off, but the rest of the body from the head to the heels is eaten.

Another source of worm infestation is the giving of raw meat, particularly the entrails of animals, to dogs.

Probably the great reason for tapeworm infestation amongst Europeans is the general lack of an efficient system of meat inspection, and the greatest source of infestation in the domestic animals is the presence of natives and native dogs scattered throughout the country. When it is pointed out that the most prevalent verminous infections that we have in meat-producing animals—measles in pigs and cattle—are both caused by tapeworms inhabiting the intestines of man, no further proof need be adduced to prove this contention. The preventive measures to be adopted are dealt with under the chapters dealing with these diseases.

The Trematodes, or short flat worms, whilst living their adult life mostly in the larger mammals, spend the earlier stages of their existence in aquatic creatures or in the water itself. This knowledge leads to a consideration of the water-supply of farm animals, probably one of the most important considerations facing the farming industry of South Africa. Not only for the reason of verminous infection, but for others as well, and not only trematode worms, but round-worms and other pests, find water a favourable medium for their existence. The water that is most favourable is stagnant water, such as is found in vleis and pans, in surface catchment dams, and in pools and rivers that remain stagnant from one rainy season to another. Water in running rivers and from springs is not so great a source of danger, but undoubtedly the safest and the best water for stock is that obtained from under ground by means of wells and boreholes, and from which the eggs of worms and other harmful matters have been filtered by mother earth.

In addition to the flukes and amphistome worms found in stagnant water, it is also the habitat of leeches and the bilharzia parasite, a protozoon that has caused sad trouble to many young South Africans, as well as affecting cattle. The question of

mosquitoes and their larvæ living in such waters and the diseases they transmit does not enter here, but must not be overlooked.

The Nematodes, or round-worms, are present in great numbers in South Africa, and the broad lines of precautionary measures to adopt in dealing with these pests may be said to lie in avoiding grazing stock upon naturally moist, wet pastures, particularly in the summer season of the year, and resting land for eighteen months or two years that has become infested with worms of a particular class of stock, to starve the worms out and so kill them. That is to say, if sheep infected with wireworm have been grazing on a pasture, keep sheep off it for the time mentioned, and graze it with mature cattle or other animals.

Medicinal Treatment.—The medicinal treatment for worms has been dealt with under the diseases caused by these pests, therefore here it is only intended to deal with the subject broadly.

Probably the best all-round preventive medicinal worm treatment for farm animals in South Africa is to allow them access to a lick into the composition of which salt and lime freely enter. The following may be recommended for the purpose:

One bag of salt, 15 pounds sulphate of iron, 15 pounds sulphur, and 125 pounds of slaked lime. The iron should be dissolved in 2 gallons of water, and the ingredients mixed together.

It is not necessary to adhere exactly to this formula—in fact, a change to simple salt and lime may be advantageous at times, or the sulphur may be left out.

Probably sulphate of copper (bluestone) is a better vermifuge than sulphate of iron; it is rather more harsh in its action on animals with a single stomach, such as the horse, and is liable to cause pain and colic, but it can be used with more freedom in animals with capacious stomachs, such as ruminants. Another ingredient that is frequently mixed in licks is arsenic, and as this is near at hand practically all over South Africa in the form of Cooper's sheep-dipping powder, it is in this form that it usually enters into the composition of the lick; but as arsenite is now produced and used on a large scale in South Africa, it is probable that this will replace the use of the powder. The following is the prescription for a lick containing these ingredients:

Sulphate of copper 1 part, Cooper's dip 1 part, slaked lime 4 parts, sulphur 4 parts, salt 40 parts.

On the whole, I am rather partial to the use of arsenic for animals in South Africa, for the reason that it not only assists in expelling worms, but it improves the appetite and probably exerts beneficial effects on the blood, which usually harbours one

or more organisms deleterious to the general health, particularly when animals are run down or weakened, as they are frequently when infested with worms.

Horses.—One of the best known and most frequently used treatments for worms in horses is 1½ or 2 ounces of turpentine mixed with a pint of raw linseed oil, and given to the animal after fasting. This is one of the oldest treatments. I have known it since a boy, and have seen most beneficial results follow its use, but I have also seen a small percentage of deaths from inflammation of the stomach and bowels follow its administration; therefore at the present time I am a bit dubious about recommending it.

A treatment that I favour is giving a powder composed of tartar emetic 1 ounce, powdered sulphate of iron 1½ ounces, gentian 1 ounce, liquorice ½ ounce; mix and divide into six powders, and give one every morning in a small quantity of bran-mash. On the seventh day give a physic ball or a pint of raw linseed oil. To the foregoing powders areca-nut is sometimes added, 1 dram to each powder, and also santonin, 10 grains to each powder.

Cattle, Sheep, and Dogs.—The treatment of the different worms in these animals will be dealt with under the diseases caused by the several species of worms.

Pigs.—The administration of medicine to pigs for worms or any other trouble is somewhat of the nature of a ticklish business, and the best way to give it is in the food. In the case of young pigs suffering from worms ¼ grain of santonin and 2 grains of areca powder for each pound of body weight may be given in a little milk after the animal has been fasted for about six hours. For larger animals 100 to 200 pounds weight 8 grains of santonin and 5 grains of calomel may be given in food. Sulphate of iron in doses of 1 dram for similar sized pigs each morning for five mornings is also beneficial. Dissolve the iron in water before mixing with the food.

Poultry.—Poultry may be dosed in the same way by mixing the worm medicine with their food, but worm pills are also used, 5 to 10 grains of areca-nut being a good vermifuge.

Common Worms.

Amongst the most frequently met with worms in the principal domesticated mammals, mention may be made of the following:

Horse, Mule, and Donkey.

Worms.	*Where Found.*
Tænia perfoliata	Intestines, but very rarely found.
Tænia mamillana	,, ,,
Tænia plicata	,, ,,
Flat-Worms	Rarely found.
Round-Worms :	
Spiroptera megastoma	Gullet and stomach.
Spiroptera microstoma	,, ,,
Ascaris megalocephala	Intestines.
Oxyuris curvula	,,
Sclerostomum equinum	Intestines and bloodvessels.
Sclerostomum tetracanthum	Intestines.
Filaria equina	Peritoneum.
Strongylus arnfeldi	Bronchial tubes.

The Ox.

Tapeworms :	
Tænia expansa	Intestines.
Tænia alba	,,
Tænia planissima	,,
Cysticercus tenuicollis	Peritoneum and liver.
Cysticercus bovis	Muscles.
Cœnurus cerebralis	Brain.
Flat-Worms :	
Distomum hepaticum	Liver.
Distomum lanceolatum	,,
Amphistomum conicum	Stomach.
Round Worms :	
Strongylus contortus	Stomach.
Strongylus convolutus	,,
Strongylus gracilis	,,
Strongylus ventricosus	Intestines.
Strongylus micrurus	Bronchial tubes.
Ascaris vitulorum	Intestines.
Œsophagostomum	,,
Œsophagostomum inflatum	,,
Anchylostomum	,,
Anchylostomum radiatum	,,
Trichocephalus affinis	,,
Filaria cervina	Peritoneum.

Sheep.

Tapeworms (*Twelve Species*) :	
Tænia or monieza	Intestines.
Tænia expansa	,,
Tænia planissima	,,
Tænia alba	,,
Stilesia centripunctata	Intestines and liver.
Cysticercus tenuicollis	Peritoneum and liver
Cœnurus cerebralis	Brain.

SHEEP (*continued*).

Worms.	*Where Found.*
Flat-Worms :	
Distomum hepaticum	Liver.
Distomum lanceolatum	,,
Amphistomum conicum	Stomach.
Round-Worms :	
Strongylus contortus	Stomach.
Strongylus cervicornis	,,
Strongylus gracilis	,,
Strongylus filicollis	Intestines.
Strongylus ventricosus	,,
Strongylus filaria	Bronchial tubes.
Ascaris ovis	Intestines.
Œsophagostomum venulosum	,,
Œsophagostomum columbianum	,,
Sclerostomum hypostomum	,,
Anchylostomum cernum	,,

PIG.

Tapeworms :	
Cysticercus cellulosæ	Muscles.
Cysticercus tenuicollis	Peritoneum and liver.
Cysticercus echinococci	,, ,,
Round-Worms :	
Spiroptera strongylina	Stomach.
Gnathostomum hispidum	,,
Echinorhynchus gigas	Intestines.
Ascaris suis	,,
Œsophagostomum dentatum	,,
Trichocephalus crenatus	,,
Trichina spiralis	Intestines and muscle.
Strongylus paradoxus	Bronchial tubes.
Stephanarus dentatus	Peritoneum.

DOG.

Tapeworms :	
Tænia serrata	Intestines.
Tænia marginata	,,
Tænia krabbei	,,
Tænia cœnurus	,,
Tænia serialis	,,
Tænia echinococcus	,,
Tænia cucumerina	,,
Round-Worms :	
Spiroptera sanguinolenta	Stomach.
Ascaris mystax	Intestines.
Ascaris marginata	,,
Anchylostomum trigonocephalum	,,
Strongylus vasorum	Bloodvessels.

The worms comprise a vast collection of animals which do not form a very natural zoological class; they contain:

1. **Hirudines (the Leeches).**—These are present to a considerable extent in South Africa, but do not greatly interest the

veterinarian. They frequently attack men in the legs who wade in the water wild-duck shooting, and at times are taken in by animals, the horse and cattle particularly, when drinking water from pans in which they exist. The leeches attach themselves inside the mouth, on the cheeks, and bottom of the tongue.

2. **Helminths,** which are divided into two classes: Platyhelminths, Nemathelminths.

Platyhelminths.—These have bodies which are generally flat, and comprise three orders: Cestoda, Trematoda, and Turbellaria. The first two only are limited to a parasitic existence. Nearly all the species are hermaphrodite.

Nemathelminths.—These are thread or round worms in which the sexes are nearly always separate, and comprise two orders: Acanthocephala, Nematoda.

Cestoda (*Tapeworms*).—These are ribbon-shaped, nearly always segmented, and provided at one extremity with organs of fixation (suckers or hooks); they have no digestive apparatus. In the adult state they live in the intestines of the higher animals, but before that period they undergo a certain number of metamorphoses and migrations which are often effected in the most diverse organs of different hosts.

They are divided into a small number of families, of which only two furnish parasites to mammals and birds. These are the Bothriocephalidæ and Tæniidæ.

Bothriocephalidæ.—This family has for its type the genus Bothriocephalus. They are tapeworms, the head of which is destitute of hooks and provided with only two suckers. The majority of the numerous species live in fishes, though some are found in carnivora and domesticated birds, as well as in man. The life history is incomplete; the larvæ have been found in pike, turbot, perch, etc., and the adults in men in most countries on the continent of Europe, but are rarely found in Great Britain.

Tæniidæ.—This family is almost reduced to the genus Tænia, which comprises more than four hundred species. It is unknown how many of these are found in South Africa, but certainly a goodly number. The Tænia are characterised by their head, which is always furnished with four suckers, between which is sometimes found a depression, at other times a prominence, which may or may not be contractile, nude, or armed with hooks. The segments or joints have nearly always their genital opening on the margin.

The Tænia have been divided into three or more groups, but

as the mode and phases of development of only a few are known, no practical purpose will be served by attempting to follow such classification.

MEASLES IN PIGS (CYSTICERCUS CELLULOSÆ)

This is perhaps the commonest of all the tapeworm affections in South Africa, and is caused by the larval stage of the tapeworm, *Tænia solium*, of man.

History.—The history of tapeworms and bladder-worms is lost in antiquity, and measles of the pig seems to have inspired Moses, the law-maker of the Jews, when the flesh of that animal was prohibited for use as food. Aristotle described the disease with remarkable precision. Redi, in 1683, seems to have been the first, however, to suspect the animal character of the measle; but it was not until 1760 that Pallas wrote his opinion that the bladder and tapeworms were related to each other.

Van Beneden was the first to prove by experiment that pigs could be infected with measles by making them swallow the eggs of tapeworms.

On the other hand, Kuchenmeister, in 1854, first experimentally established that human beings could be infected with the tapeworms *Tænia solium* by feeding them on pig's flesh containing the bladders. Kuchenmeister fed criminals condemned to death on measly pork. After death he examined the contents of their bowels, and found the heads of young tapeworms clinging to the intestinal membrane. Since then several enthusiastic students of pathology have purposely and experimentally infected themselves with tapeworms by eating measly pork.

Nature.—The bladder-worms or measles that are found in the flesh of pigs are scientifically known as *Cysticercus cellulosæ*, and are the larval stage of the particular tapeworm in man known as the *Tænia solium*. Each little bladder contains the head of a young tapeworm, which is called a scolex. These heads, being only about $\frac{1}{30}$ inch in size, are too small to be satisfactorily examined with the naked eye, but under the microscope it is seen that the head is exactly like that of a full-grown tapeworm, only smaller: there are the four suckers and a circle of hooklets which help the scolex to become attached to the intestinal membrane, which they do when they are introduced into the alimentary canal of man by means of uncooked or undercooked pork. As soon as the scolex has become attached it commences to grow, and segment after segment is developed

from its posterior portion; therefore it follows that the segments farthest away from the head are the oldest and most mature. As long as the head remains in the intestines, it continues to originate fresh segments. Each segment is a being in itself; it contains ovaries in which the egg becomes fertilised and develops. As the posterior segments become ripe and full of eggs they are cast off from the worm. They are passed out from the bowel with the excrement, and by this they frequently gain entrance to the stomach of the pig, and there and in the intestines the eggs hatch out; the embryo penetrate the intestinal wall and gain entrance to the blood circulation, by which they are transported throughout the system. They become arrested in the small vessels of the muscles and organs, where they find a favourable situation for their further development. After a period of about three months they are fully developed and ready to commence another life cycle if eaten by man, and cycle after cycle can go on for an indefinite period. Very rarely a change in the life cycle has been known to take place, in that a human host of a tapeworm has infected himself with tapeworm eggs, and in such cases the measles become located in his muscles similar to that which occurs in the pig. The disease has also been recorded in dogs on several occasions.

Symptoms.—There are no recognisable ante-mortem symptoms, and the disease cannot be diagnosed during life, except that in a small number of cases these little bladders may be seen on the under surface of the tongue; but a great number of pigs are badly infected with measles without having the bladders located in that site.

Post-mortem Appearances.—The bladders or cysts are grey in colour, and are from a kaffir-corn to a small pea in size. They lie between the muscle fibres, and vary in number from one or two to many hundreds. The bladders contain a clear fluid and a small nodule, which contains the head of the future tapeworm. In old-standing cases the bladders degenerate and their contents become thick and yellowish or greenish in colour, and in about six months the bladders shrivel up and become gritty. The commonest sites for the bladder-worms are the tongue and the muscles of the cheek and neck. They are frequently found in the various organs, but, excepting in the heart, this is not common.

Treatment.—There is no known curative method. The treatment should be of a preventive nature.

Owing to slaughter throughout the greater part of South

Africa being allowed on privately owned premises, and no comprehensive system of meat inspection being practised, it is impossible to state to what extent the disease is present; but in the Bulawayo abattoirs, where a system of inspection is in force, about 6 per cent. of pigs are annually condemned for measles. These pigs are almost entirely reared by European farmers. In the case of native-owned pigs the percentage would be considerably higher.

The first step in preventive treatment appears in a thorough system of meat inspection and the proper disposal of infected carcases. By freezing such carcases and keeping them so for a sufficient length of time the cysts will be destroyed or die, but facilities for this treatment do not exist at many places. Even if they do, the question of it being an economical method arises owing to the length of time required. Liquid salting in brine for several weeks will also destroy the cysts. What usually happens is that at places where meats are inspected and where facilities exist the carcase is rendered down for its grease; at others it is simply seized and destroyed. At private slaughter-houses the manner of the disposal of the carcase will depend upon the proprietor, and knowing human nature to be what it is, little doubt exists that in some cases visible measles are cut off and flesh that probably contains measles (unseen) goes for human food. As compared with the number of Europeans affected with tapeworm, the percentage of natives is greater, which gives colour to the suspicion that much of this measly pork is disposed of to natives.

The next step in preventive treatment or eradication is educating the people to the means by which the disease is spread. The disease is spread to the greatest extent first by the habit of natives, and to a lesser extent of Europeans, defæcating upon the veld. This practice, of course, can never be entirely eliminated, but it can be lessened to a considerable extent by pig-keepers and others providing suitable latrine accommodation near to their homesteads. Secondly, it is spread by allowing pigs to roam about the homestead and indulge in their coprophagous habits. When pigs are grazed they should be confined in a pig-proof paddock, one they cannot get out of, and in which natives are allowed to go as little as possible. It is not only by their coprophagous habits that pigs become infected; when the eggs leave the human body they possess a considerable amount of resistance, and may be carried by rains and floods and contaminate the water-supply, so that is another matter that

has to be taken into consideration when planning preventive measures.

Some pig-keepers, to avoid the animals contracting measles, keep their pigs constantly confined in a sty, and never allow them to be at large. This system is in the great majority of cases entirely successful, but occasionally it fails in that one or more animals are found to be affected, and difficulty is found to account for the manner by which they become infected. When it is remembered—provided there are people about the place infected with *Tænia solium*—how easy it is for the drinking water or the food of the pig to become contaminated with the eggs of this worm, the existence of such odd cases may be explained. There is another point that is frequently overlooked, and that is that, although it is usual for the mature segments (proglottides) containing the eggs to be expelled from the human host at the time of defæcation, they also leave the body at other times without any inconvenience further than a slight irritation at the anus to their host. I have seen this happen; therefore it is possible for a person to drop one or more such segments in any place even without knowledge of the fact that it has happened.

This leads to consideration of a further line of preventive treatment, and that is the expulsion of the tapeworm from the body of its human host. Both European and native carriers of tapeworms are anxious for them to be expelled from their bodies, and usually will willingly submit to medicinal treatment if it is available, which it should be in all cases. The treatment that I have found most successful is fasting the patient for twenty-four hours, then giving up to 2 fluid ounces of castor oil mixed with one dram of extract of male fern. This is a full dose, and should only be given to big, strong natives; a smaller dose of both ingredients should be given in proportion to the strength of the patient.

Another preventive measure is the thorough cooking of pork, as, when this is done, the heat destroys the cyst, but a difficulty exists in getting the inside parts of the joint sufficiently heated to do so; further, there are people so fond of pork that they cannot resist the temptation to partake of it when it is placed in front of them, and they will take the risk of becoming infected with tapeworm sooner than forgo the meal, therefore a system of meat inspection which would prevent measly pork getting into the hands of the cook is the better way.

Measles in pigs under Roman-Dutch law is a latent disease, therefore the seller is held responsible, and the killers can, and

usually do, claim the total purchase price back from him. In a few places a system of insurance at the abattoirs has been suggested or tried, but it is not very successful, as it is unfair in its incidence upon the careful and cleanly pig-keeper.

MEASLES IN THE OX (CYSTICERCUS BOVIS)

Measles in the ox is produced by the larval form of the *Tænia saginata*, or the unarmed tapeworm of man. The head of this worm has four suckers and a central pit, a point which distinguishes it from *Tænia solium*.

History.—Although the existence of the unarmed tænia of man was noted nearly two centuries ago, its origin was only recognised at a relatively recent period. The *Cysticercus bovis* appears to have been seen for the first time by Judas in 1854 in Algeria. In 1819 Knox witnessed at the Cape a real epidemic of tapeworm infestation among English soldiers, who for the most part were fed on the flesh of overdriven cattle.

Nature.—Next to measles in pigs, this is the commonest form of tapeworm infestation in South Africa, for the reason as stated regarding pigs. Comprehensive statistics are not available as to the frequency of its occurrence, but in Bulawayo it averages about 2 per cent., and perhaps this may be regarded as an approximate average for the whole country.

The bladder-worms are similar to those seen in the pig, but usually slightly smaller, and are frequently met with in the muscles of the jaw, the neck, and in the heart.

The symptoms, post-mortem appearances, and treatment as described for the pig apply equally in the case of measles in the ox. It has been concluded that the cysts in the ox do not live so long as in the case of the pig; that after seven or eight months they dry up and calcify; and Oliver states that in India the cysts disappear from cattle a few months after they receive well water.

This reference to well water refers, of course, to the freedom of such water from tapeworm eggs in contradistinction to surface water, and the profusion of eggs in a single tapeworm furnishes an explanation as to the frequency of ox measles, and the readiness with which infestation occurs is due to the resistance of the eggs to causes of destruction. Davaine says he has kept them for fifteen months in water more or less pure without their losing their vitality. Cobbold held that a man affected with tapeworm expels about 400 proglottides (mature segments) per month, and

that each of these may contain 30,000 eggs, so that a total is reached of more than 140,000,000 expelled by one person in the course of a year. Whether the foregoing be regarded as an outside number or not, it gives an idea of the opportunities for cattle to contract measles, even although a number of such eggs are destroyed by various causes.

Cattle in India are reported to have coprophagous habits similar to pigs. I have heard of them having such a habit in South Africa, but I do not think it exists to any great extent.

CYSTICERCUS TENUICOLLIS

This is the cystic form of *Tænia marginata* of the dog. It is one of the largest of the cysticerci, and known to butchers by the name of " water-ball " or bladder, and varies in size from a pigeon's to a hen's egg; the neck of the cyst is long and thin.

Nature.—The *Tænia marginata* is the largest tapeworm found in the dog. The head is scarcely wider than the neck. The development of this tapeworm is relatively slow; it takes from four to five months after the ingestion of eggs by the dog before the worm reaches maturity and the mature segments containing eggs become detached. The cystic form of this worm is usually met with on the peritoneum (outside covering of the bowels), and more rarely on the pleura of the lungs and heart sac of various animals, more especially ruminants, the ox, sheep, and goat being common hosts; it is sometimes met with in the pig, and has been found in the camel and reindeer.

The eggs of the tapeworm reach the intestines of these animals with the food and water, and experiments have shown that the majority of the embryos, when hatched out, reach the abdominal cavity by way of the liver; their passage through the liver usually occurs without any serious disturbance to health, but cases have been recorded of it causing death in lambs and kids. The parasites tunnel through the liver, leaving a trail behind them usually filled with a blood-clot. On about the tenth day after the ingestion of the egg the larvæ are through the liver and commencing to develop into cysts, and in about seven or eight months they are completely developed and ready to continue the life cycle if swallowed by a dog.

Treatment.—Treatment is of a preventive nature, and consists in not allowing dogs to have access to viscera infected with this cyst; but the treatment that will appeal most to stockowners is

the prevention of the sheep becoming infected. This can best be done by treating dogs for tapeworms and ridding their grazing grounds of innumerable worm-infested native curs.

CYSTICERCUS OVIS

Cysticercus ovis is the larval form of the *Tænia ovis* of the dog. The cysts closely resemble those found in pig measles; they are usually but few in number, and are most often encountered in the heart and diaphragm. They have also been found in the goat. This disease has been observed in England, and to a greater extent in America. I am not aware that it has so far been discovered in South Africa.

Amongst other forms of cysticerci, mention may be made of the following:

Cysticercus pisiformis in hares and rabbits is the larval form of the *Tænia serrata* of dogs.

Cysticercus tarandi in reindeer is the larval form of the *Tænia Krabbei* of dogs.

Cysticercus fasciolaris in rats and mice is the larval form of the *Tænia crassicollis* of the cat.

GID IN SHEEP AND GOATS

Synonyms.—Sturdy, Turnsick, Malkop.

This is a fairly common disease in South Africa, which is caused by the *Cœnurus cerebralis*, and is the cystic stage of the tapeworm of the dog known as the *Tænia cœnurus*.

History.—The disease occurs in all countries. Its nature was for a long time unknown, but it was supposed to be an apoplexy, a dropsy of the brain, etc. Leske, in 1780, was the first to recognise the watery bladder as a cystic worm always found in animals affected with gid; but it was not until 1853 that exact notions of the true nature of the disease began to prevail, when Kuchenmeister experimented with it. Similar experiments have been conducted since by many other investigators, which have established in the firmest manner the relationship of the cyst in the brain of the ruminant to the tapeworm in the dog.

Nature.—The *Tænia cœnurus* is almost exclusively a parasite of ruminants, but it is more frequently found in the sheep and the goat than in other animals; it has been met with in the antelope and the horse. It principally affects young animals, lambs, kids, and calves; it is rarely found in calves, but it causes considerable

losses in goats, particularly in Angora kids, and a still greater loss in sheep.

The life history of the parasite is similar to the others already described. When a lamb receives the mature segments or eggs of the *Tænia cœnurus* the shells of the eggs are dissolved in its stomach and the embryos are set free; these possess six hooks, by means of which they pass through the walls of the stomach or intestine and probably get into the blood-stream, and are carried by it to various parts of the body. Fortunately many of the parasites are destroyed during transmission by the blood-stream, and comparatively few arrive at maturity—surprisingly few when the number of eggs scattered by dogs on the pasture is considered. Those which arrive in the nerve centres are almost the only ones to pursue their development. On arrival in the cranial cavity, the embryos lose their hooks and are transformed into vesicles, which in the course of about three months become fully developed bladders, usually about the size of a pigeon's egg, but they may be as large as a hen's egg. These bladders consist of a thin wall containing a clear watery fluid; attached to the wall are a large but variable number of small white nodules about the size of a mustard seed. These are the heads of the future tapeworms. If the whole bladder is swallowed by a dog, each of these heads is capable of developing into a distinct tapeworm, which in its turn develops in the intestines of the dog, and in due course gives off mature segments full of eggs; these are scattered over the pastures by the dog, and during grazing are picked up by the sheep, and so the life cycle again commences.

The disease is one for the wet season of the year, and is more prevalent in damp, low-lying, marshy ground. This is due, as has been proved by experiment, to sunshine and air desiccating the mature segments and eggs and rendering them inert. This takes fourteen days in Europe; in parts of South Africa it would no doubt occur quicker; on the other hand, it has been proved that they may remain infective for three months when kept in damp grass.

The eggs are disseminated over the pasture by the rains, and washed into stagnant pools and dams at which the animals drink.

A matter of importance is whether any of the wild carnivora act as host to the tapeworm *Tænia cœnurus;* so far as experience goes in Europe, this is not so.

It has been held, and it appears reasonable, that animals that

bite close to the ground when grazing are more liable to become infected than others that do not crop so closely.

Symptoms.—The symptoms presented will depend to a great extent upon the number, the age, and the situation of the bladders in the cranial cavity or brain. It has been observed that if several worms lodge themselves in the cranial cavity at one time, the first symptoms of gid may be observed from eight to twenty days after the eggs have been ingested; but this is rarely observed, and, if present, the symptoms pass away and the real symptoms of gid are not present until the bladder-worms are from four to six months old. During this time an attentive observer may recognise at different times, such as during stormy weather, certain signs indicating pressure upon the brain.

The bladder, being situated in the cranial cavity, by pressure upon the parts of the brain regulating movement in different sets of muscles, naturally governs the automatic movements of the animal, and determines whether the affected animal will walk in a circle to the right or the left, and whether the head will be carried high or low.

The commonest symptoms are: the animal appears dull and stupid, the face shows a vacant stare, the ears lop, and the eyes appear unequal in size. The animal walks in circles, which usually become smaller and smaller, until at last, pivoting on itself, it falls down. As a rule the animal usually turns towards the same side on which the brain is compressed, but on rare occasions, when two or more cysts are present in different portions of the brain, it moves sometimes to the right, sometimes to the left. In other cases it bores forward, pressing its head against anything that comes in the way. Other cases frequently fall backwards; in other cases paralysis of the hind limbs will be present; and in somewhat rare cases the eyes may pirouette in the sockets.

There may be an association of several of these movements, manifested by repeated attacks in the course of the day, and the disease may last from a few weeks to a few months if slaughter does not intervene to prevent death from extreme exhaustion.

Treatment.—In the great majority of cases it is not worth while attempting curative treatment, the better plan being, as soon as a case is diagnosed, to hand the animal over to the butcher while it is still useful for food purposes, and before it becomes emaciated.

In certain instances, such as in the case of pure-bred stud animals, it may be desirable to attempt a cure by a surgical

operation; this may be successful in a certain number of cases, particularly if there happens to be only one cyst in the brain, and that situated to the front and at the surface. The first difficulty encountered is to locate the site of the cyst. The way the animal holds its head, and whether it circles in its movements to the left or right, may give assistance in locating the spot, the cyst being usually on the same side of the brain that is held lower or that the animal circles to. The head should next be clipped, and then, if the point of the finger is pressed over the surface, owing to the thinness of the bone over the cyst, the signs of pain caused by the pressure will serve as a guide. When the spot where it is determined to operate has been decided upon, the skin should be thoroughly cleansed by washing with disinfectant in the water, then the skin should be incised in the form of a cross over the spot, the corners turned back, and the subcutaneous tissue cut away to expose the bone. The instrument that is then required is a trephine; a drill or a trocar may be used, but a trephine cutting a circle $\frac{1}{2}$ inch in diameter is better. When the opening has been made at the right spot, the cyst projects into it; it should then be punctured; an ordinary hypodermic syringe to draw out the fluid answers the purpose very well. When this has been done, seize the cyst with a pair of forceps and twist it round and round, getting it into a rope, and gradually pulling it out whilst twisting it. The wound can then be treated as an ordinary wound, stitched up, and painted over with iodine tincture.

Preventive treatment is the one indicated for the disease, but owing to the number of native dogs in this country it is often a difficult one to attempt. Where possible, all dogs upon the farm should be subjected to a course of treatment. Fast the dog for twenty-four hours, and then administer $\frac{1}{2}$ to 1 dram of extract of male fern mixed with 1 to 2 ounces of castor oil according to size of dog; place the bottle containing the dose in warm water to heat it, and so render it more fluid; tie the dog up; collect the tapeworms passed and burn them. Areca-nut (powdered) in doses of 1 to 2 drams mixed with milk after fasting the dog is also good for expelling tapeworms.

On farms where the disease is present graze the sheep on the highest and driest portions during the wet season.

The heads of all sheep suffering from this disease should be burnt; it is a safer method of disposal than burial. By the latter method dogs may disinter and eat them, and so repeat the life cycle of the tapeworm.

ECHINOCOCCOSIS

The *Echinococcus veterinorum* and *Echinococcus polymorphus* represent the cystic stage of the *Tænia echinococcus*, which lives principally in the intestines of the dog. This worm often escapes observation, owing to its small size; it only comprises three or four segments, and appears as yellowish filaments distended at one end. The life cycle is similar to that of other tapeworms: when the eggs reach the digestive tract of the herbivora or man the embryo is set free; this may encyst in any organ of the body, but the liver and lungs are its favourite sites.

The cysts are commonly called hydatids, and are met with in very varied hosts. They are most frequent in ruminants and the pig, but are also found in man, and have been found in a great number of other animals such as cat, dog, rabbit, monkey, horse, sheep, goat, antelope, elephant, kangaroo, etc.

The development of the hydatid is very slow, and each mother cyst is capable of giving rise to daughter cysts; therefore, when the liver becomes involved, it is usually considerably augmented in volume and weight, sometimes tenfold.

Treatment.—The only treatment is of a preventive nature, by dosing dogs for worms and having as few about the farm as possible.

Stilesia centripunctata is a tapeworm of very common occurrence in the livers and intestines of sheep and goats in Rhodesia and Bechuanaland—in fact, it may be said to be the exception to find a liver free from this parasite. It is also found in the liver of buck such as the stembuck, duiker, water-buck, and roan antelope.

It is a delicate worm inhabiting the bile-ducts of the liver; the segments are broader than long, and its life history is unknown.

Other Tapeworms.—A great number of other tapeworms exist of which the life history is unknown. The horse has three, cattle have a similar number, sheep harbour twelve, the cat four, the fowl nine, the duck eight, and other birds also possess tapeworms.

The pig so far has never yet been found to harbour a tapeworm, and may be regarded as the freest of all animals in this respect; on the other hand, the dog puts a heavy tax upon mankind in return for his friendship and usefulness by harbouring no less than thirteen or fourteen different species of tapeworm. In addition to those already described belonging to this animal,

brief mention may be made of two others. The *Tænia serialis* of dogs passes its cystic stage in small rodents, particularly rabbits, and the *Tænia cucumerina*, with segments shaped like a melon seed (cucumis), finds its intermediary host in the dog flea, so that when a dog is busily engaged in "fleaing" he may be catching tapeworms at the same time.

TREMATODA

The Trematoda are platyhelminthes, the bodies of which are nude, soft, and non-segmented, and furnished with one or more suckers; they have a digestive canal with a single opening, and no anus. Those which concern us live as parasites in the interior of the body of their hosts, and belong to the suborder Distomea. They possess at most two suckers, of which the anterior is oval. With rare exceptions they are hermaphrodite, and lay eggs which, when they arrive in moist places, generally in water, give rise to embryos which usually seek out some aquatic creature and undergo one stage of their development. After a time this host is left and another similar aquatic host is obtained for another stage of the life cycle, and from this or with their host they find their way to the stomach of the third host, and the life cycle is completed.

SCHISTOSOMIASIS

Bilharzia, as it is commonly called, the disease caused by the *Schistosomum hæmatobium*, may be said not to interest the veterinarian, as it is a disease affecting man; but even for that reason it is worth knowing something about, and further that probably in the course of time similar diseases will be found to affect the domestic animals to a greater extent than are at present known.

History.—There is every reason to suppose that this disease has existed from the very earliest days. Evidences of such parasitism have been said to have been found in the mummies of Egypt, and the Mosaic law in regard to the danger of snails apparently confirms the existence of the disease in the time of the Pharaohs.

The name *Bilharzia hæmatobia* (*distomum*) or *Schistosomum hæmatobium* was given by Cobbold to a trematode worm discovered in 1851 by Bilharz at Cairo in the portal vein of man. Since that time the disease has been found to exist in many parts of the world—Mauritius, India, Venezuela, and probably in all parts of Africa. It is fairly common in parts of the Union and Rhodesia.

An analogous worm was found in 1876 in Egypt by Sonsino in the portal vein of a bull, and subsequently in the sheep.

Nature.—So far three different worms have been found affecting man. These are *S. japonicum*, which causes disease in the large bowel (colon), the rectum, and mesenteric nodes; the worms inhabit the small bloodvessels (arterioles) of the intestinal wall, and the eggs are spineless. The *S. mansoni* causes disease in the back bowel (rectum), and the eggs possess cruel-looking lateral spines. The *S. hæmatobium* causes urinary disease, the seat of the worms being in the bloodvessels in the walls of the bladder. The egg possesses spines at the ends, and when these make their way through the wall of the bladder into its interior, to be passed out with the urine, they cause bleeding, and the blood is likewise voided with the urine.

Until recently nothing was known of the life of this interesting parasite outside the human body, or of the manner in which it gained entrance to the body. Now it is known that the egg (ovum) containing a living larva leaves the body in the urine, and after coming in contact with fresh water, ruptures the enveloping capsule and escapes, swimming about actively by means of numerous ciliary projections. Later the larva or miracidium finds an intermediate host in the body of a snail. In the Union this has been found by Dr. F. G. Cawston, Medical Officer of Health at Krugersdorp, to be the *Isidora schakoi*; in other countries similar snails play this rôle.

In the liver of the snail it develops into great numbers of long cylindrical bodies, the sporocysts and daughter sporocsyts. Within each of these a cercaria develops and escapes into the water. This has the power of penetrating the skin of man and other animals, and so gains entrance to the bloodvessels and lymphatics. By this route it finally reaches the liver, where it is ready to begin a new cycle of development into the flat-worm.

After about six weeks the worms reach maturity; they are bisexual, unite in pairs, and descend against the current of blood into the mesenteric and vesical veins, the *Bilharzia* variety choosing the latter route, thus gaining entrance to the bladder wall, where the eggs are deposited. Just how this is accomplished is still somewhat obscure, but it seems natural to suppose that the suckers of the male play an important part, as they do in other animal parasites. The eggs being deposited in the bladder wall, the life cycle of the parasite is completed.

In addition to the parasite in the cercarial stage of its life cycle being able to enter the body through the unbroken skin,

as they undoubtedly do in numbers when people, particularly young ones, are bathing in fresh-water pools, a number are taken into the system by drinking such water containing them, and it is therefore a matter of concern to know the freedom from this parasite of the water-supply of a household, or even of a town supply, and in the case of the latter, if it is contaminated, whether the filtration is efficient.

The parasite and the snails, according to Dr. Cawston's investigations, are more frequently present in stagnant pools than in running rivers, and are absent in water holding a fair quantity of lime in solution.

Treatment.—Preventive treatment lies in draining the ground where possible, and liming it or applying lime direct to the pools; one bag of lime to every 1,000 cubic yards of water will appreciably diminish the snails found in water-holes. Snails are air-breathers, and cannot live in water devoid of air, so that a thin coating of paraffin on the surface of a stagnant pool, such as is used for the destruction of mosquitoes, would kill them. Professor Ross's larvicide consists of 300 gallons crude carbolic acid, 200 pounds resin, and 30 pounds caustic soda. One part of this mixture, which is well boiled in preparing, is added to 5,000 parts of water. It will kill any larvæ or cercariæ in less than five minutes.

AMPHISTOMA

This is a trematode worm that is frequently found in the rumen of cattle and sheep in the Union and in Rhodesia. I have come across it in the stomach of cattle in great numbers, without it doing apparently any great harm to its host. In cattle it is about ½ inch in length, ovoid in shape, a rosy fleshy colour, and slightly curved on one side. In sheep it is somewhat smaller. It is usually erroneously regarded as a "bot" by farmers and others.

The intermediary host in the Union has been identified by Dr. Cawston as the fresh-water snail, *Isidora schakoi*. The ruminants take the parasite into their system by eating damp, moist grass upon which the cercariæ have encysted. There appear to be several different species of adult worms, and probably more than one species of fresh-water snail may act as their respective intermediary host.

FASCIOLIASIS OR DISTOMIASIS

Synonyms.—Rot, Liver Rot, Fluke.

History.—The distomes of the liver have been recognised for a very long time, but the first mention of them is due to Jean de Brie in 1379. In 1547 Gabucinus spoke of worms resembling pumpkin seeds inhabiting the liver of sheep and goats, and in course of time other observers commented upon them in a similar manner. From 1880 to 1882 Thomas in New Zealand conducted investigations on behalf of the Royal Society of England, and worked out the evolution of these parasites.

They are world-wide in occurrence, and are particularly parasites of ruminants, affecting both domesticated and wild species. They are found besides in several other domestic and wild animals —the horse, ass, pig, elephant, rabbit—and even in man. In South Africa, as in other parts of the world, they affect the sheep and the ox to a greater extent than any other domestic animal.

Nature of the Disease.—The causal parasite is called a fluke, from its resemblance to the flat-fish known as a flounder or sole. There have been several species of flukes determined, but the two that affect domesticated ruminants more than others are the *Distomum hepaticum* (*Fasciola hepatica*) and *D. lanceolatum*. The former is the more important, being larger, commoner, and existing in greater numbers. It is found in the liver and bile-ducts of cattle, buffaloes, sheep, goats, antelopes, and camels; less frequently also in the liver of the horse, ass, pig, elephant, and rabbit, and even in man. The latter is frequently found along with the former in the bile-ducts of ruminants, and has also been found in the dog, cat, and man. The *D. hepaticum* has the shape of a privet leaf or small sole; it is of a pale brown colour, and is about 1 inch or 1⅓ inches in length, and in breadth about half its own length.

At one end is a narrow projecting portion; this is the head, and at its tip is placed the mouth, in the middle of which there is a smaller sucker by which the fluke can attach itself. At the point where the head joins the body and on the under surface there is a second sucker, called the ventral sucker, hence the name of distoma, two-mouthed or two-suckered. In South Africa the existing flukes have not been exhaustively determined. In Rhodesia I have observed flukes in sheep answering in size and description to *D. hepaticum*. I have also seen much larger

flukes in cattle that appeared to be different to any I have read descriptions of.

The intermediary host of the fluke is a small fresh-water snail, the *Limnæus truncatulus* being the most frequent carrier of the parasite; this snail is present to a limited extent in South Africa, but Cawston (Krugersdorp), who has investigated the subject to a certain extent, is of the opinion that the *L. natalensis* is the intermediary host of the parasite in the Union. This seems most possible, as the disease is present in parts of the world where *L. truncatulus* is not found.

The following description of the *L. truncatulus* may be taken as a general rough outline for snails likely to act as intermediary hosts for flukes: A fresh-water snail with a brown spiral shell. It is very small, its shell never reaching more than ½ inch in length, whilst it is usually much smaller—less than ¼ inch. It is often difficult to find, owing to its minute size and its habit of wandering from the water. That the snails can live in moist ground, quite away from any quantity of water, for considerable periods has been proved by the fact that they have kept alive for eleven weeks on moist grass and moss, even when infected with the larval form of the liver fluke.

The following is the life history of the fluke: Starting with the adult parasite attached by its suckers to the inside of a biliary duct, the following stages occur: The fluke scatters large numbers—30,000 to 40,000—of eggs into the duct. These pass with the bile into the stomach and intestines of the host, where they may hatch out. Usually they do not, but are excreted with the fæces, and so find their way to the ground. It is possible for these eggs to remain dormant for some time, but those which are fortunate in reaching pools of water hatch out under the combined influence of warmth and moisture, the resulting offspring being known as ciliated embryos. For the next stage this embryo swims about in search of a suitable intermediary host, a particular fresh-water snail. If the embryo does not find the suitable snail it dies; if it is successful, it bores its way into the snail and locates at the bottom of the pulmonary chamber, where it transforms into a sporocyst, which grows and forms within itself bodies known as rediæ, and these in time break loose from the mother sporocyst, and in due course attach themselves to the liver of the snail. Next the rediæ themselves give rise to new forms possessing a tail and known as cercariæ. These cercariæ, after leaving the rediæ, bore through the snail until they reach water, through which they swim, and at length

attach themselves to blades of grass. Here they develop into a cyst or spore, and await to be swallowed by an animal. In the stomach of the animal this cyst becomes dissolved, and the young fluke emerges and pursues its course to the liver, where it grows and becomes mature, and thus recommences the life history its parent before had done.

It must not be concluded that it is only grass immediately around the permanent pools of water that have these cysts upon their leaves. It is natural for the snail to travel fair distances from such pools, and naturally the cercariæ leave the small body when they are ready to do so, irrespective of the place the snail may be in at the time. Providing the surrounding conditions are favourable, such as moist grass with a certain amount of water on or around it, the cercariæ attach to the grass and encyst. In this connection the point that the cercariæ in the majority of cases attach low down on the grass is worth noting, as it is held that this fact, coupled with sheep grazing closer to the ground than cattle, accounts for the former animals becoming worse infected than the latter.

The number of parasites that a single fluke can give rise to is really astounding, and the number of cercariæ descended from a single fluke egg cannot be fewer than 200, and is sometimes much larger; for the rediæ forming the second generation, instead of producing cercariæ, may, and perhaps as a rule do, give rise to another generation of daughter rediæ resembling themselves, and these then produce cercariæ. When this is the case, a single fluke egg may give rise to more than a thousand cercariæ. It will be seen, therefore, that not only does the race of the liver fluke multiply and increase abundantly in its vertebrate host by producing myriads of eggs, but there is a further and great increase of the forms within the snail. If only the greatest degree of ordinary increase were reached, a single fluke might give rise to many million descendants in the next generation of liver flukes proper, but fortunately for stockowners the chances are enormously against such disastrous increase.

Temperature exerts a considerable influence on both the fluke and the snail. A temperature of about 74° to 78° F. is the most favourable for the growth of the embryo within the egg; therefore it follows that the disease is most active in the summer-time. The snail buries itself in mud or soil in the winter, and owing to the cold no embryos are hatched out at that time.

So far as South Africa is concerned, temperature probably plays a more important part in the south in such places as the

Bontebok Flats, the Stormberg and the Sneeuwberg ranges of mountains, where the disease is very prevalent, than it does in the north such as Rhodesia. In that country moisture exerts a greater influence, although, owing to the rains coming in the summer, the disease is present at the same season of the year. The disease is one of wet, marshy, water-logged places. Ground of a clayey nature that has water standing on its surface instead of sinking in is said to be "rotting." In Southern Rhodesia the disease exists in parts of Mashonaland only, but in Northern Rhodesia, owing to the principal rivers overflowing their banks and inundating the surrounding country, the conditions are particularly favourable for the disease, and it affects cattle to a considerable extent.

Rain plays another part in washing the eggs that have been scattered over the veld by infected animals, and carrying them to stagnant pools and other places where the conditions for carrying out the life cycle of the parasite are particularly favourable. If the cysts of the fluke that are attached to grass steme are not swallowed within a few weeks they perish. After such a cyst has been swallowed by an animal, in from six weeks or a trifle longer the parasites become mature and begin to produce eggs.

This knowledge of the seasonal occurrences and the localities infected with fluke provides one means of preventing the disease by only grazing such lands in the winter.

Symptoms in Sheep.—The general appearance of sheep when suffering from fluke in the liver differs but little from the unthrifty and emaciated condition of sheep which are infested with large numbers of other internal parasites such as the *Strongylus contortus*, or wireworm, found in the fourth stomach, tapeworms, and round-worms, which infest the intestines. One of the most characteristic symptoms of fluke in the liver, when the animal is seriously affected, is the dropsical condition of the cavities and tissues of the body. This is indicated by the appearance of a dropsical swelling, or bag, which forms under the skin and hangs down from the throat and between the branches of the lower jaw. This is called "dikkop" by the farmers. This dropsical, bag-like swelling is more pronounced in sheep that are affected with fluke in the liver than it is in any other form of poverty and emaciation arising from the presence of other internal parasites or any other debilitating cause.

It will be observed, also, that the contents of this dropsical

bag increases considerably during the day when the sheep's head is held down in the act of grazing, and it correspondingly decreases towards morning, owing to the sheep lying down during the night with its head held up while ruminating. This is easily understood, because the contents of this bag consists of a clear watery serum which gravitates towards the head from the chest during the day, and drains back again to the chest during the night, being assisted considerably by the movements of the lower jaw in the act of rumination.

The first effect of the invasion of the bile-ducts by young flukes is a tendency to fatten very rapidly. This effect has been made use of by owners by placing sheep intended for slaughter upon fluke-infested pastures. It is believed to be due to ths stimulating action of the young flukes on the bile-ducts, increasing the biliary secretion and promoting the assimilation of the food. This period lasts usually one to two months; however, if a large number of flukes have invaded the bile-ducts it may be shortened, because as they increase in size they irritate and distend the ducts and cause considerable thickening of the fibrous covering of the walls. The irritation gradually extends to the liver substance adjoining the affected bile-ducts, and causes congestion and a slow form of inflammatory action, which gives rise to an increase in the fibrous framework of the liver, which presses upon and largely replaces the liver cells and correspondingly impairs their secreting function.

It is this destruction of the liver cells and the consequent impairment of their secreting function that leads to the serious interruption of the processes of digestion, assimilation, and the healthy functions of the organs of the body generally, which is indicated by the rapid manner in which the affected animal falls off in condition. The sheep looks dull, the eyes lose their brightness, the conjunctiva becomes pale, presenting a puffy, watery appearance. The wool is dry and harsh, clapped close to the body, and is easily pulled out; there is rapid emaciation, and a general anæmic and dropsical condition. The backbone becomes sharp and prominent—"razor-backed"—and the abdomen becomes pendulous, diarrhœa sets in, followed by exhaustion and death. The rapidity with which these symptoms become developed will depend much upon the number of flukes which have invaded the liver and the local conditions, such as the character of the weather and the quality of the veld. If the weather is bleak and cold and the veld dry and innutritious, these symptoms will follow each other in rapid succession. If,

on the contrary, the veld is good and the other conditions are favourable, they will develop more gradually.

Post-mortem Appearances.—The pathological lesions present in a case of fluke will largely depend upon the extent of the infection and the length of time that the flukes have existed in the liver. In the earlier stages there is little marked change visible; the liver is enlarged and slightly uneven, with hard, cirrhotic patches and bluish-white streaks on its surface. There is slight distension of the bile-ducts with flukes. In more advanced cases the liver becomes hard and irregularly knotted or corrugated, the bile-ducts become greatly enlarged and their walls much thickened and hardened, and their lumen distended with flukes crowded together. In very advanced cases the true cellular structure of the liver is largely displaced by the thickened fibrous tissue which forms the external coat of the bile-ducts and the bloodvessels, and also constitutes the minute framework of the liver. The liver is greatly altered in shape and considerably diminished in size, and so hard that it cuts like cartilage, conveying a crisp, gritty sensation to the knife.

In cases of long standing in which the animal has survived the acute effects of the flukes the larger bile-ducts stand out like solid fibrous cords, most of the flukes having escaped, and the lumen of the ducts becomes filled with fibrous tissue.

The general lesions are: The muscular tissue of the body is softer and paler in colour than natural, and in advanced cases it is flabby and watery looking. The abdominal cavity contains a variable quantity of a clear watery serum, and the abdominal organs also present a blanched and watery appearance. In fact, the whole of the fat and cellular tissue throughout the body is infiltrated with serous fluid. The chest and heart-bag also contain a variable quantity of a similar fluid, often mistaken for heart-water. The flesh does not stiffen after being slaughtered and dressed.

Cattle present similar symptoms and post-mortem appearances to the sheep, but on account of their greater power of resistance the disease is less severe. When cattle were imported from North-Western Rhodesia into Southern Rhodesia it was rare to find an ox that was slaughtered for beef that was not either actively infected with fluke or whose liver did not show the enlarged, thickened bile-ducts as big as a man's thumb that had been caused by fluke. In cattle the dropsical swelling between the jaws and along the windpipe is not so frequently seen as in sheep, but the digestive disorders are present.

Treatment.—In considering measures to adopt for the prevention of this disease, the first thing that naturally occurs to one is the drainage of the infected pastures, and I think there is little doubt that this is the best permanent method that can be adopted. Whether it can be economically done by such simple means as ploughing a few furrows that will wear with the rains into deeper drains, or whether it can be done at all, are matters that can only be decided by those concerned, each case on its merits.

Ploughing and cultivating the land, keeping animals off it, except in the winter when the snail is dormant, and many, if not all, the cysts are dead, fencing off the water-holes and the surrounding moist vleis, and allowing the animals only well water to drink, are among the methods that will occur to those who are unfortunate in having fluky pastures on their farms, and there is little doubt that the adoption of one or a combination of these measures will be found of benefit in many cases.

As a preventive measure next in importance to drainage comes the use of salt. In countries where it is cheap this is spread on the land at the rate of 400 pounds to the acre. This would be a too expensive process for many parts of South Africa, but when it is stated that as low as a 1 per cent. salt solution will kill cercariæ in less than half an hour, it can be realised that benefit is derived from dissolving a small quantity of salt in the animals' drinking water or giving them the use of a salt lick, by its action upon the parasites in the animals' stomach before they have reached the liver.

Lime is also most deleterious upon the parasite and its intermediary host, the snail; a $\frac{3}{4}$ per cent. solution of it will kill both the embryo and the snail as well, therefore liming the land should be adopted in all cases when the cost of the material is not prohibitive.

A farming practice that is most praiseworthy is to provide stock with a lick. This is frequently composed of 40 pounds each of coarse salt and lime with 2 pounds of powdered sulphate of iron. Some people also add arsenic in the shape of half to one packet of Cooper's sheep-dipping powder. Such a lick is good in keeping several of the internal parasites down. One way to prepare it is, after thoroughly mixing the ingredients together in a dry state, to moisten them, and place them in a box or boxes of suitable size, then tamp down and allow to dry out and harden.

The following lick is highly spoken of as a preventive of fluke and other internal parasites:

Common salt	6 parts.
Powdered slaked lime	1 part.
Sulphur	1 ,,
Sulphate of iron	1 ,,

Mix thoroughly and keep continually before the stock.

With regard to the curative treatment of the disease, it should be remembered that the ox is a hardier animal than the sheep, and is better able to withstand the damage caused by flukes in the liver. Secondly, that the amount of constitutional disturbance and inflammation set up in the liver is in proportion to the number of flukes present; therefore the first line of action indicated is to remove the infected animals to dry, fluke-free pasture.

Until the last few years no curative medicinal treatment was claimed for fluke, but recently Raillet, Moussa, and Henry claim to have effected cures by the use of ethereal extract of male fern. The doses to be used are as follows: For an ox, 10 to 25 grammes according to weight; for a sheep, 3 to 5 grammes according to weight.

The remedy is given mixed with 10 c.c. of non-purgative oil for a sheep and 20 c.c. for an ox or cow; one dose to be given in the morning two hours before the animal is fed, five doses in all to be given on five consecutive mornings. From the published results of the experiments the drug apparently acted only on the large variety of fluke (*D. hepaticum*), but even if it only does that, by reducing the number of flukes present it would considerably assist in recovery, and is worthy of a trial in South Africa; a probable drawback to its employment for ordinary stock will be its cost. The drug must be used with care in the matter of the quantity given, as it is liable to set up brain symptoms and giddiness; and it would be advisable to use the smallest doses recommended for animals in this country owing to the small size of the animals.

The greatest remedy for the debility caused by fluke or any other internal parasite is good nourishing food, and plenty of it.

Having removed the animals from the infected pastures to good grazing free of the parasite, or having supplemented their grazing by supplying them with other food, and supposing that no medical treatment has been employed, what usually happens is that the severely smitten animals die off fairly early, while the

others will gradually recover, as no additions are made to the number of the flukes in the liver after the removal of the stock from the tainted ground; further than that, a gradual decrease is going on by the flukes dying. The length of the life of a fluke has been held to be about twelve months or longer. Hutcheon produced apparently conclusive evidence that flukes lived as long as four years in sheep in Cape Colony; but this does not alter the fact that if animals have survived for some months after removal from a fluke-infested pasture, the number of flukes they will then harbour will usually not seriously interfere with their health; and if the changes set up in the liver originally have not led to serious chronic changes in that organ, a return to practically unimpaired health may be expected.

One matter that may seriously disturb the landowner is the danger of infecting new pasture by moving fluke-infested animals on to it; this danger will depend upon the nature of the pasture and whether it is sufficiently moist to harbour the snail. The chances are that the snail and the fluke are already there, and it follows that there is little or no risk in moving animals to fluke-free pasture. Such movements have been made in innumerable instances in South Africa without tainting the pasture following. I have had personal experience of hundreds of fluke-infested cattle being grazed in the vicinity of Bulawayo without the land becoming fluke-infested.

NEMATHELMINTHES OR ROUND-WORMS

THE round-worms comprise two orders, Acanthocephala and Nematoda.

ACANTHOCEPHALA.

Species of this order are found in the rabbit, duck, swan, etc., but the best-known species is found in the pig, wild and domesticated, and sometimes in the striped hyena; this is known as the *Echinorhynchus gigas*. The giant *Echinorhynchus* is a round-worm of a milky white tint, wrinkled transversely, and larger in front than behind. The head is almost globular, and possessed of five or six rows of hooks, with which it attaches itself into the mucous membrane of its host, inhabiting mostly the small bowel. The male worm is about 2 to 3 inches long, and the female 9 to 12 inches. When these worms are in any great number in the bowels they cause marked disturbance in nutrition and frequently cause the death of young pigs. The life history of the worm is not altogether settled; the eggs are said to be taken up by the larva (the white worm found in manure) of the ordinary May-bug, (*Melolontha vulgaris*), and are conveyed to the intestines of the pig by that animal eating the worms. It would appear that other Arthropodes (invertebrates) may act as intermediary hosts.

NEMATODA.

The nematoda have a digestive canal generally complete with two openings. They are elongated, frequently slender, and even filiform. Their number is considerable, and they are found in all the organs of the domesticated animals with the exception of the bones. They may be white, yellow, brown red, or marbled in colour. The respiration is exclusively cutaneous. The sexes are always separate—at least, in the species that interest the veterinarian. The male is habitually smaller than the female. They have been divided into a great number of families, seven of which furnish the parasites of the digestive apparatus in the

domesticated animals. These are Ascaridæ, Oxyuridæ, Strongylidæ, Trichotrachelidæ, Filariidæ, Gnathostomidæ, and Angiostomidæ.

Ascaridæ.

All the ascarides live in the intestines, and especially in the small intestine of various vertebrata. This family contains some of the commonest species of round-worms met with in the domesticated animals. The genera are Ascaris and Heterakis. Almost every species of the domesticated mammals has a corresponding species of Ascaris. Among the domesticated animals poultry alone harbour species of Heterakis. The life history of these worms is but imperfectly understood, some observers contending that there is no radical change in the egg outside the body of its host until it again enters the body of another host of the same species as the first, whilst other observers contend that an intermediary host, an invertebrate, is necessary. It is held that oxygen is necessary for the embryo to develop inside the egg, hence the development of the embryos can occur only outside of the animal body. Eggs which contain embryos appear to be endowed with a particular power of resistance, since they retain their viability for six months while kept in a suitable medium such as desiccated horse manure.

The following worms occur in domesticated animals:

Ascaris megalocephala.—This is the large, round, rigid, spindle-shaped, yellowish-white worm that is so frequently seen in the intestines, particularly the small bowels of the horse, mule, and donkey. Frequently only a few worms are present, which have given no evidence of their presence during life; at other times they are present in hundreds, and several pounds' weight of them have been recorded as being found in one animal. The largest specimens (females) of this worm will measure 8 to 10 inches. Animals of all ages are infected, but the worms are more prevalent in young animals and in the horse than in either the mule or donkey.

Ascaris vitulorum.—This worm lives in the small bowel of calves; it is rarely found in adult cattle. It has a reddish-white, transparent body, and greatly resembles the worm found in the horse, and is nearly as large.

Ascaris ovis.—A yellowish-white worm that has only been found on rare occasions in the bowels of the sheep.

Ascaris suis and Ascaris lumbricoides.—The ascaride of the pig (*suis*) and the one of man (*lumbricoides*) bear such a close resemblance to each other—in fact, they are indistinguishable in shape

and make—that several authorities consider they both belong to the same species. The ascaride of the pig, although widely distributed throughout the world, is perhaps not very common except in certain localities.

The experience of medical men and veterinarians who have practised amongst men and animals in Asia has led them to the belief that an interchange of the parasites between man and the pig takes place by the eggs of the worms in man being spread with manure among the lettuces and other salad plants; on the other hand, pigs are allowed to roam and indulge their coprophagous habits, and many facilities are provided for them to do so. We in South Africa, from our knowledge of the methods of Asiatic gardeners, can appreciate this view of the matter.

F. H. Stewart, of the Indian Medical Service, has had special opportunities of tracing these worms in man and pigs. His researches, however, led him to the conclusion that the eggs of the ascarides are not taken direct, but must be first ingested by rats or mice, and that in these intermediate hosts the embryos come away, undergoing transformations and migrations (perhaps to the same extent as the fluke-worm of sheep) until they find their ultimate host in man or the pig, their excrement having fouled the foods of either.

La Clinica Veterinaria gives a summary of investigations made by various scientists, and among them Ransom and Foster. These observers have reached the conclusion that an intermediate host is not necessary, but that the eggs when ingested open in the digestive tube; that the embryos do not at once take up their residence in the gut, but wander into other organs, particularly the liver, lungs, and spleen. Here, perhaps, may be found the explanation of some pigs carrying a good number of worms without detriment to their health or perceptible hindrance to growth and development, while others of the same farrow or under identical conditions of feeding and management waste, become hidebound and unthrifty, and, in a word, show evidence of being "wormy." The wandering of parasites in the tissues and the giving off of waste (toxins), even in the bowel, is known to be one of the causes of injury by parasites. How much more, then, if migrations are carried on into the delicate organs? Some young children and young animals seem to offer special attraction to parasites of this class, and it may well be that in these or others the originally selected home is not liked, and the wandering habit is taken up by larger numbers in particular individuals than in others.

In subjects of experimentation the worms have been found to have reached the lungs in a week or less. They reach the stomach by way of the back of the mouth (pharynx) and down the gullet. This migration has been demonstrated in pigs and rats as well as mice. In this way they gain the intestine for the second time, and in a suitable host (man or pig) settle down until they have attained maturity. If they do not like the host they let themselves be carried out in the ordinary way. This is what happens when they find themselves landed in the rat or mouse, but it may well be that individuals of the natural host (man or pig) prove distasteful to them by reason of excess of sulphuretted hydrogen or carbonic acid gas in the canal. This probably accounts for the fact that horses that have been living on dry, hard food in the stable for a lengthy time, when given a change and supplied with green food, such as barley and lucerne, frequently expel a number of ascaride worms; this is particularly noticeable in animals prone to flatus.

It seems likely, from the experiments made by the authorities quoted, that many cases of pneumonia are caused by invasion of the lungs by the ascarides during the wandering period. One of the experimental subjects who was compelled to ingest a large number of eggs died of pneumonia in a week.

We are aware that fatal results often occur in infants and pigs infested by these worms.

Ascaris marginata and Ascaris mystax.—The ascaride of the dog (*marginata*) greatly resembles that of the cat (*mystax*), and they are thought to be of the same species. They are prevalent in young animals in the bowels; when they are present in great numbers they frequently get into the stomach, and when this occurs kittens will be observed to vomit them up.

Ascaridæ in Poultry.—These worms (*Heteraki*) are very common in poultry and world-wide in occurrence, one particular species of worm being more prevalent in one country than in another. In England the commonest species is the *Heterakis perspicillum*; in Australia the *H. compressa*. In South Africa the species, so far as I am aware, have not been determined. A great number of species of these worms exist throughout the world, attacking practically all classes of the domesticated feathered animals.

The life history of the parasite is supposed to be direct, by the egg passing from host to host of the same species, but it seems equally possible that in certain instances—such as might occur in the case of fowls and the ants—an intermediary host will

be found to play a not unimportant part in the life cycle of the parasite.

As a cause of ill-doing these worms are rarely suspected by the poultry keeper, but those who make post-mortem examinations have an idea of the frequency and extent of the loss suffered by their depredations. Hardly an adult fowl can be found without any intestinal parasites. A few may do no harm, but crowding of birds upon a limited area provides the conditions for rapid multiplication of these pests.

The disease known as " going light " is often due to the extensive invasion by these worms. The cæca or blind gut will be sure to contain them, if present at all, so that the poultryman whose birds do not respond to generous feeding should satisfy himself as to their presence or absence by opening that portion of the bowel and washing it free of ingesta. It is a yellowish-white worm, in length from 1 to 2 inches. The female, as with most ascarides, is larger than the male. Their mode of reproduction favours their rapid dissemination in a flock, eggs of microscopic size being produced in great numbers and passed with the droppings upon the ground.

The medical treatment of single birds is seldom a profitable business, and it is to prevention that we must chiefly look.

Changing the site as often as possible is advised; but, without killing off the worms, this may be only a greater distribution of the parasites. Runs, yards, and frequented places should be washed down with liquid disinfectants, and when dry again dusted over with lime.

The doses of medicine usually prescribed for fowls are out of all proportion to their weight as compared with quadrupeds, and a teaspoonful (dram or 60 drops) of spirit of turpentine seems a large one for a creature of 2 or 3 pounds weight, but in practice it proves desirable. It should be mixed with a teaspoonful of olive oil and given on an empty crop, and the bird confined in a clean-swept place in order to observe the results.

Oxyuridæ or Whipworms.

The best-known representative of this family is the

Oxyuris curvula or the Curved Oxyuris.—This round-worm is a parasite in the intestines, especially in the large bowels of the horse, mule, and donkey. The female of this worm (commonly known in England as the " maw-worm ") is almost exclusively met with. It is from 1½ to 2 inches long; the head is curved in

a similar manner to the handle of a walking-stick, and the body tapers out into a thin tail. Whilst these worms may be found in all portions of the large bowel, it is held that the mature female worm holds itself by its tail near the anus of its host, and whilst there deposits its eggs; these pass out with a sticky white substance and adhere around and below the anus. Under the influence of free oxygen, which is present in this situation, the embryos develop within the eggs in one to three days. Owing to drying up, this substance drops off, carrying with it eggs which may infect the manure or the food; the embryos remain alive quite a long time. After ingestion by a suitable host the embryo escapes from the egg, and about three months elapse before the parasite arrives at maturity.

The symptom of the presence of this worm, in addition to the deposit of the white substance around the anus, is an itchiness of the part, the animal rubbing its tail against the wall or other object, causing a roughening of the hair at the root of the tail.

The treatment frequently advised is enemas of vinegar and soap and water, coupled with worm treatment; but I have found the latter sufficient, and perhaps this is the line of treatment indicated as by it the immature as well as the mature worms are expelled.

Other known Oxyuridæ are:

Oxyuris mastigodes.—The long-tailed oxyuris. This has been held by Railliet to be only an abnormal form of *O. curvula*. The female is from 5 to 6 inches long.

Oxyuris vermicularis.—This worm is frequently found in the rectum of man. The female is from $3\frac{1}{2}$ to 4 inches long.

Oxyuris ambiguus.—Occurs in the rabbit and the hare.

Oxyuris compar.—Occurs in the cat.

Strongylidæ.

The Strongylidæ comprise numerous genera which are divided into three tribes: (1) Eustrongylines, (2) Strongylines, (3) Sclerostomines.

The genera are: Œsophagostomum, Syngamus, Globocephalus, Sclerostomum, Stephanurus, Anchylostomum, Ollulanus, Hystrichis, Physaloptera, Strongylus. Eustrongylus, Stephanurus, and Syngamus are not parasites of the digestive canal.

Eustrongylines

The only representative of this tribe in the domesticated animals is the

Eustrongylus gigas.—This is the giant Eustrongyle. It is the largest of the Nematodes, and is a parasite of the kidneys; it is met with in man, horse, ox, and dog. It is much more frequent in the carnivora, especially in the dog, but it is only exceptionally met with.

It is generally a blood-red worm, slightly tapering at the extremities, and finely striped transversely. The male is from 5 to 16 inches long and $\frac{1}{4}$ inch wide. The female is from 8 to 40 inches long, and from $\frac{1}{4}$ to $\frac{1}{2}$ inch broad.

It is not common for more than one worm to be present; this entirely destroys the kidney, the worm completely filling same. It frequently migrates from the kidney, and is then found in other parts of the body.

Strongylus

There are eight known species of Strongylus which live in the air passages of various domesticated mammals. Other species are found in the intestines of ruminants and the rabbit. The best-known species in South Africa is the

Stomach Wireworm (*Strongylus or Hæmonchus contortus*).—This parasite, which is prevalent amongst sheep and goats in South Africa, causes more losses among small stock than any other of the internal parasites of domesticated animals. It infests the fourth stomach of sheep and goats principally, but may be found also in cattle and various wild ruminants.

Its distinguishing name, *contortus*, is given to it because the female has a red and white spiral appearance, like a barber's pole. This arises from the arrangement of its ovarian tubes, which are rolled round the intestine to form regular loops, the intestine being straight and of a pinkish colour, which, when the worms become older, changes to a brownish hue and becomes more distinct. It subsists on the blood it abstracts from the mucous membrane, and to this it owes its colour. It seems that small particles of the mucous membrane are also ingested by it. The worms are small, the average size being: male about $\frac{3}{4}$ inch and female about 1 inch long, and about as thick as an ordinary pin; therefore their presence may be overlooked by the casual observer, although they may be present in thousands. The

presence of these worms in the stomach results in the irritation of the mucous membrane, causing disturbance of digestion and resulting malnutrition; moreover, the worm is a blood-sucker, and the blood-sucking habit results in anæmia of the affected animal, causing it to become feeble and thin. More especially are lambs and young sheep affected seriously, but often fully-grown sheep, although badly infested, provided the veld is in good order, may show no apparent symptom of the disease.

Symptoms.—The general symptom is a pernicious anæmia shown by loss of flesh, weakness, debility, thirst, and diarrhœa. The mucous membranes of the eyes and mouth are pale and bloodless, and in the advanced stages a dropsical effusion appears under the skin of the lower jaw and abdomen.

Life History.—This has been worked out by the Veterinary Research Division of the Union, and is as follows:

In the adult stage of the wireworms two sexes, male and female, are recognised, which can be distinguished by their respective size, the female being larger than the male, and possessing a peculiar protuberance in the hind third of its length.

Adults.—Males and females only live in the stomach of sheep and other ruminants (cattle, goats, antelopes), and in particular in the abomasum or fourth stomach. Here they are found at all times of the year in more or less great numbers. The fact has been established that a sheep that has been infected once with wireworms and is not treated will remain infected to a more or less degree for at least a year, and probably longer. This fact is of fundamental importance, because it is the infected sheep that spreads the worm infection from place to place. The females are fertilised in the stomach, and then the egg-laying begins, and continues practically all the year round, but is more pronounced in some seasons than in others—*e.g.*, less in winter than in summer.

Eggs.—The eggs leave the stomach and pass through the intestines, and are deposited with the fæces. The number of eggs that can be passed by a sheep of healthy appearance has been calculated to amount to three million per day, but it is likely that in badly infected sheep it is much greater.

First Larva.—Eggs only hatch outside the body. Inside the eggshell the embryo develops within nineteen hours under the most favourable conditions. The hatching process requires an optimum condition of moisture and temperature of the ambient medium. This medium is the pellets of the sheep. After leaving the eggshell the worm is in its first larval stage, which is completed under most favourable conditions at the end of

twenty-four hours. During this stage the larva remains closely attached to the medium in which it has been growing. It forms a new skin, called the first skin. The body detaches itself from its old skin; this opens itself at the anterior end, the larva wriggles out, and the second stage begins.

Second Larva.—This larva is very lively, feeds freely on the medium, and grows rapidly. It again undergoes a further change, and forms a new skin. The old one becomes detached and envelops the body completely, which meanwhile has changed to the third stage.

Eggs and first larval stages cannot infect a sheep, but the larva that has undergone the second stage can do so; it is called the mature larval stage. It is this stage which is endowed with longevity; before this will be dealt with the cycle will further be outlined.

Third Larva.—The mature larva reaches the stomach of a sheep, and on entering it casts its second skin, and thus the third stage continues its evolution, ending in the formation of a new skin, the third one, and casting it again. The ensuing evolution taking place inside a host is now called the parasitic part of the cycle. The young parasitic larva that comes out of the third skin is now in its fourth stage. It bores itself into the surface of the mucous membrane of the stomach, and in doing so causes a slight effusion of blood, which coagulates, and it is within this coagulation that the larval worm is found.

Fourth Larva.—It forms here its fourth skin. The differentiation of the sexes takes place in the fourth stage. The period of the third and fourth stage requires in adult sheep about a fortnight for completion.

Adults.—The fifth stage, which is entered into after breaking of the fourth skin, is that of adults, males and females. Maturity is reached after a further period of about fifteen days. Only then, after fertilisation has taken place, the process of egg-laying begins again. It requires thus at least one month from the day of entry of a mature wireworm larva until new eggs are voided. This is of fundamental importance, because, as will be seen later, it determines the period within which a sheep should be dosed at least once.

Thus repeating the life cycle of the wireworm, we distinguish the following stages: Non-parasitic stages are the eggs, first and second stages; parasitic are third and fourth larval stages and the adults (males and females).

The Mature Larva.—The mature larval stage is of extreme

importance from a practical point of view, inasmuch as it is the infective stage, and for this purpose is endowed with a number of qualities that enable it to reach the host. The first quality is its longevity under suitable conditions. These are moisture and absence of sunlight, when it will live for a period of up to one year. The second quality is that of migrating. As soon as the new skin is formed and the old one is detached, the wandering of the young worm begins; the tendency is always upward, and the young worm crawls on to the grass in order to reach its destination, the stomach. This migration is, however, subjected to a number of influences. It only takes place when the surroundings of the worms are moist; it cannot crawl on a dry surface. Thus the most suitable conditions are the rainy seasons, or when heavy dews have fallen, or when the grass is in a vlei. This migration is further influenced by light. Direct sunshine is not appreciated by the young worm; and as the light gets stronger and stronger, the young worms immediately hide and return to the ground. Thus the favourable periods of migration are the nights and the cloudy and rainy days, when there is a complete absence of sunlight; diffuse daylight does not interfere with this migration. Direct sunlight kills the worm. Dryness is badly resisted by them. When at the same time exposed to light the larval worm retires into moist soil, where it remains alive.

Practical Deduction.—For the practice the following deductions can be made: Mature larvæ are only able to infect sheep under conditions of moisture—*i.e.*, in the rainy seasons and in the vleis. They are absent from the pasture during the winter when the grass is dry; they may be found in the vleis, being able to exist in water.

Diagnosis by Means of Culture.—A practical demonstration to show the development of larvæ and their migration can be carried out by every farmer by placing sheep pellets into a jam glass, screwing down the lid, placing the glass in a cupboard, and watching events. From the fourth day, provided sufficient moisture is present, which has deposited itself as fine dew on the inside of the glass, the larvæ begin to migrate and form white slimy tracts ramifying over the surface. When the glass is now exposed to light it can be seen that the worms return to the fæces, and so long as these are wet will enter them again. Sour fæces are unsuitable for this purpose. This method will enable a farmer to find out to what extent his sheep are infected with wireworms before they show the ill effect of the infection.

Conclusion.—The above notes bring out clearly two points: (1) A living sheep maintains the infection and spreads it so long as adult wireworms are in its stomach; (2) an infected pasture remains infected for at least a year, and all sheep grazing in it during this time become reinfected.

Eradication of the Worms.—In order to suppress the wireworm plague it is necessary to kill the wireworms in the stomach of the sheep and clear the farm of mature larvæ. The first condition can be carried out with certainty provided the remedy is made use of as recommended by the Union Division of Veterinary Research. The second one could be carried out by starving the larvæ through keeping away for at least one year all animals that act as hosts—sheep, goats, cattle, antelopes. This is difficult or impossible. The alternative suggestion, therefore, is to have the mature larvæ picked up by sheep and to kill them in the stomach of the sheep. Since the parasitic worm requires one month to reach sexual maturity before it again lays eggs, the dosing must take place at least within a month, and must be repeated during the months of rainy seasons. After the lapse of one year the mature larvæ which have not reached their host have practically all died, and during this time no eggs have been scattered about; hence, after the lapse of one year a farm should be clean of wireworms. It should also be clear from these notes that a remedy cannot be thought of that keeps wireworms away from the stomach.

Analogy with Tick Eradication.—The principle underlying the above recommendations is that applied in the eradication of ticks. Ticks cannot be prevented from attaching themselves to stock; therefore stock will collect them, and the bath in which the stock are dipped will kill them; likewise the sheep collect the wireworms, which are then killed by means of the dosing. In the case of ticks short intervals are necessary to do so; in the case of sheep an interval of a month between the two operations will be sufficient. As by means of continued dipping the tick plague is reduced to small danger, so will be the result with the systematic dosing of wireworms.

Treatment.—The Division has perfected a remedy that has been used with excellent results in very many cases; but to ensure that only the best quality ingredients are used and in the correct proportions, the prescription has not been made public, but is prepared and distributed by the Research Division at cost price to stockowners needing it.

The remedy is primarily designed for the eradication of wire-

worms (*Hæmonchus contortus*) in ruminants, particularly in sheep, but it is incidentally effective against tapeworms.

The remedy is supplied in standard tins each containing sufficient powder for single dosing of 100 or double dosing of 50 adult sheep. The size of the dose varies with the age of the sheep, so that the standard quantity of 100 adult doses provides 125 doses for two-tooth sheep, 166 doses for lambs between six and ten months old, 250 doses for lambs between four and six months, or 312 doses for lambs from two to four months old.

The remedy is issued at the following prices and places, with full instructions as to its use, which should be strictly observed: Dosing spoons, 1s. 6d. each, or 7s. 6d. per set of five, for dosing animals of various ages. Dosing bowls, 1s. 6d. each. Remedy, 1s. per standard tin, containing sufficient powder for single dosing of 100 adult sheep. Post or rail free to any part of the Union. These articles can only be forwarded on receipt of remittance to cover cost, or by rail c.o.d. to the nearest railway station where this system is in operation.

All communications from the Transvaal and Orange Free State should be addressed to the Director of Veterinary Research, P.O. Box 593, Pretoria, and Veterinary Research Laboratories, Pretoria (Telephone 245, Pretoria; Telegraphic address, "Microbe," Pretoria. Parcels by rail, Pretoria North Station).

All communications from Natal should be addressed to the Senior Veterinary Research Officer, P.O. Box 405, Pietermaritzburg (Telephone 316; Telegraphic address, "Bacteria").

All communications from the Cape Province should be addressed to the Veterinary Research Officer, P.O. Box 41, Grahamstown (Telegrams, "Institute," Grahamstown).

WIREWORM OF THE OSTRICH

Synonyms.—Haarworm or Draadworm (Dutch).

The worm causing this disease was named *Trichostrongylus douglasi* in 1881 by T. Spencer Cobbold, for the reason that the material for his investigations was supplied him by an experienced and at that time well-known ostrich farmer, the Honourable Arthur Douglas, of Heatheton Towers, Grahamstown.

It is a very small worm infesting the stomach of the ostrich, the male being on an average slightly under $\frac{1}{2}$ inch long and the female slightly longer.

The life history of this worm has also been worked out by the Veterinary Research Division, and is very similar, as also are the

effects of the parasite upon its host, as those described in the *Hæmonchus contortus*. They were unable to find any remedial treatment by drugs, but were enabled to show by experiments that the solution of the wireworm problem laid in hatching the chicks in an incubator, or removing them from the nest directly they were hatched and rearing them on clean ground in a small run. Infected ground can be cleaned by removing the top 3 inches of soil and then covering it over with a similar thickness of sand, the young birds to be kept in such a run until they have reached the age when wireworms are no longer so dangerous to them. Ostriches can withstand an enormous infection of wireworm if they are well fed. Ostrich chicks do not suffer from wireworm infection even when well fed, but may succumb from larval infection—that is, before the larvæ reach the adult stage.

Strongylus cervicornis (*MacFadyean*).—This worm was found by MacFadyean in lambs in Lincolnshire, England, in 1896, where it caused a heavy mortality. It is about $\frac{2}{5}$ inch long, and the principal symptom is scouring.

Strongylus gracilis.—A similar worm also discovered by MacFaydean in cattle in England in 1894, and since then found by him in the stomachs of sheep, horses, and pigs.

Other strongyles of the stomach and intestines that have been described and named are:

Strongylus axei or *tenuissimus*, found in tumours of the stomach of the donkey.

Strongylus convolutus, found in the fourth stomach of the ox.

Strongylus filicolis, found in small intestine of sheep and goats.

Strongylus nodularis, found in the intestines of the goose.

Strongylus quadriradiatus, found in the intestines of the pigeon.

Strongylus retortæformis, found in the intestines of the rabbit.

Strongylus strigosus, found in the intestines of the rabbit.

Strongylus tenuis, found in the intestines of the goose.

Strongylus ventricoccus, found in the intestines of the ox and sheep.

Strongylus vicarius, found in the stomach of sheep.

BRONCHIAL AND PULMONARY STRONGYLUS

Of the eight known species of strongylus which live in the air passages of domesticated mammals, the following species have been observed in South Africa:

VERMINOUS BRONCHITIS OF CALVES

Synonyms.—Husk or Hoose, Worm in the Windpipe.

This was the first one of this class of disease to be observed, which was so long ago as 1744. Since that date it has been often observed and described. The disease is not very prevalent in South Africa; it occurs principally along the south-eastern and eastern districts of Cape Colony.

It is caused by the *Strongylus micrurus*, an ovoviviparous filiform worm tapering at the extremities, of which the male is about $1\frac{1}{4}$ inches long and the female 2 to $2\frac{1}{4}$ inches in length. This worm is rarely met with in older cattle.

Symptoms.—When the strongyles are not numerous, the symptoms are a slight sonorous cough, occurring in fits at varying intervals. As the worms increase the cough becomes stronger, and occurs in paroxysms, accompanied by great difficulty in breathing, the animal becoming almost suffocated. During these attacks the animal shows great distress, the breathing is quickened, the flanks heave, the head and neck are extended, the mouth is open, the tongue hanging out, and frothy saliva dribbling from the mouth. These fits of coughing may occur several times during the day, and the animal may die in one of them. The coughing causes the expulsion by the mouth and nasal cavities of mucus that may be streaked with blood, and contains single worms or masses of them, which are readily recognised, more particularly by their movements when they are put into tepid water.

When the disease progresses slowly, which it frequently does in the Union, the symptoms are milder, but the animal becomes gradually emaciated, the mucous membranes pale, the eyes sunken, the skin dry, scurfy, and very frequently infested with lice; the breath becomes fœtid, and diarrhœa is a frequent accompaniment towards the final stage.

The diagnosis of the disease is based principally on the character of the cough and its occurring in paroxysms. But the true test is the presence of worms or their embryos in the discharge from the mouth and nostrils.

Treatment. *See page* 400.

VERMINOUS BRONCHO-PNEUMONIA OF THE SHEEP AND GOAT

This disease appears to have been first observed in 1768 by Daubenton in flocks of sheep at Bourgogne. The disease exists in South Africa, principally in the Western Province of Cape

Colony. It has also been observed in sheep coming from the vicinity of, and slaughtered at, Kimberley, but it does not cause serious loss. The worm causing the disease is the *Strongylus filaria*, the male being of a brownish colour and measuring from 1 to 2½ inches in length; the female is slightly longer and white in colour. The *Strongylus rufescens*, which is frequently associated with the former in other countries, has not been observed in South Africa, although the appearances presented by the lungs frequently indicate the lesion attributed to these small strongyles.

Symptoms.—The *Strongylus filaria* of lambs occupy the lower portions of the bronchial tubes principally, but may extend up into the trachea, similar to the *Strongylus micrurus*, producing bronchitis and symptoms identical with those seen in calves. There is an acute bronchial catarrh, a strong, painful, and convulsive cough occurring in fits, and difficult respiration. These paroxysms of coughing leave the animal weak. There is a thick and abundant discharge from the nostrils, and masses of mucus are often expelled during the attacks of coughing, in which the worms or their subjects are often found.

Death arises from emaciation and debility, aggravated by difficulty of breathing. This disease may occur at any time of the year, but it is generally most pronounced and intense from midsummer to the beginning of winter. Damp, warm seasons are most favourable for the preservation of these parasites. In South Africa they are generally found where there is some undrained vlei grazing.

Treatment. *See page* 400.

VERMINOUS BRONCHITIS OF THE PIG

This disease was first noticed towards the end of the eighteenth century, and has been recorded many times since then; but in Europe it is not of such frequent occurrence or of such a grave nature as the disease in calves and lambs is. In South Africa the reverse is the case. Hutcheon states:

"The parasitic bronchitis of pigs, which is due to the *Strongylus paradoxus*, is much more common and much more serious to that industry than either of the other two forms of parasitic bronchitis. The first opportunity which I had of verifying the parasitic nature of this affection was in 1891, in the Wellington district, but since that time I have seen it over a large area of the Western Province. It is principally confined to young pigs."

The *Strongylus paradoxus* is the worm causing the disease. The male measures about ¾ inch; the female is a little longer

Symptoms.—In the first stages a cough is the only indication that the animal is infested; by-and-by the symptoms become more aggravated, and the breathing becomes distressed (called *jagziekte*). This becomes more and more pronounced until the animal dies.

Post-mortem Appearances.—In an affected animal which has been killed and bled for examination the lower portions of the lungs present a purple colour, and are semi-solid from occlusion of the bronchial tubes and collapse of the lobules surrounding them. On opening the bronchial tubes with a small knife or pair of scissors, the small round worms will be found lodged at the extreme end of the tubes. The cough in early stages is due to the irritation produced by these worms, but as the irritation increases the smaller terminal tubes of the lungs become blocked up, which curtails the breathing space and gives rise to the rapid and distressed respiratory movements termed by the farmer *jagziekte*.

Life History.—These strongyles are ovoviviparous—that is, the female worm produces eggs and embryo worms. The embryo worm is already developed within the egg. These embryos escape from their shells within the bronchial tubes; numbers of them are coughed up with mucus by the infected sheep. In this way the infection is spread over the pasture or they get into the pharynx, and are swallowed and voided with the fæces. Other known species of strongyles infecting the lung are oviparous, and in these it is only after some time has elapsed that the embryo escapes from the egg, whereas in the ovoviviparous it escapes almost as soon as the egg is deposited.

The complete life history of these worms is not known, but it is a matter of observation that they breed and develop in moist pastures and marshy places. Cobbold, who made a series of experiments with the *Strongylus micrurus*, was of the opinion that the young strongyles undergo their primary changes of development either within soft mud alone or within the bodies of larval insects and minute entomostraca (shell-fish). It is the general opinion that these strongyles pass their first phases in an intermediary host.

Investigators are also undecided as to how these worms get into the bronchial tubes of the animals. Many think that the worms are swallowed with the food, and return from the stomach up the œsophagus, and enter the respiratory organs at the opening of the larynx. Others entertain the opinion that they enter the nostrils when the animals are feeding, and then

crawl down into the bronchial tubes. The opinion most generally held is that the worms are taken into the stomach with the food, and that the minute embryos pass from the intestines through the villi into the circulation, and are by that medium conveyed to the lungs.

It may be stated that our knowledge of the life history of these parasites is very meagre. It would appear as if the larva has to go through certain stages outside the vertebrate host, as attempts to infect animals by larvæ obtained from the bronchial tubes have failed. Possibly these stages may eventually be found to be somewhat similar to those that occur in the *Hæmonchus contortus*.

With regard to the manner in which the parasite obtains entry into its host, present-day knowledge will point to ingestion as the most probable way. The question of the means by which the parasite gets from the stomach to its predilection site in the lungs is still unsolved. Many favour the idea of transportation by the blood-stream, and in support of this theory quote the case recorded by Neveu-Lemaine, who found mature specimens of *Strongylus filaria* in the windpipe of a fully developed fœtus of a mother sheep. Personally I am of the opinion that insufficient weight has been given to the power of these and similar parasites of determining and making their way unaided by the easiest and perhaps most direct route to their predilection sites.

Treatment.—This should be mainly preventive, by no means an easy matter on a farm which has once become infected, because the embryos are capable of being revivified after several months' desiccation. It would appear that moisture is necessary for the development of these embryos during their intermediary stage outside the lungs of their particular host, whether that is passed in mud or in molluscs.

When the disease is discovered amongst a flock of sheep or a herd of calves or swine, all visibly affected animals should be immediately isolated from the rest, and such affected stock should be grazed on pasture growing on a dry, porous soil, free from any moist or marshy places in which the embryos could undergo their intermediary stage of development. They should also be prevented from drinking at the same place as the healthy stock.

The healthy stock should also be kept away from all such marshy places as much as possible, leaving these to be grazed by older cattle or horses, and a supply of uncontaminated water

provided for them. Where such an arrangement is impracticable, the water should be drawn into troughs and about 1 per cent. of common salt added to it along with a little lime.

With respect to pigs, similar treatment should be carried out; all affected pigs should be placed in sties and prevented from wandering about infecting the runs. The clean pigs should also be fastened up in sties previously cleaned and disinfected, so that they may be prevented from roaming about and contracting the infection. The whole lot should be fattened off as early as possible. No pigs should be reintroduced until two years after the last of the previous lot has been cleared out. It is possible that a less period might be sufficient if proper attention were paid to the cleaning and disinfection of the sties and moist places around the homestead.

Another form of preventive treatment is indicated which I consider of considerable importance, and that is, if it is assumed that the parasite gains entrance to its host through the stomach, in which it will remain for a time, it should be destroyed whilst in that organ by putting the affected animals through a course of wireworm remedy prepared by the Veterinary Research Division.

It may be only a coincidence, and it may be something more, but frequently animals that harbour worms in the windpipe are at the same time infested with worms of a similar nature in their intestines. There may be a great number of these, as well as species of other worms, and the clearing of these out, if even it fails in preventing a further number of strongyles finding their way to the lungs, will relieve the animal and greatly assist in one of the best-known curative measures, which is supplying good nourishing food. This cannot have the desired effect on an animal whose intestines are full of worms.

Curative Treatment.—Some thirty to forty years ago a cure was often attempted by causing the infected animals to inhale various vapours, one of the favourite being the fumes of sulphur, the procedure being to put the animals in a building that was as air-tight as possible. The operator then went inside armed with a vessel containing some red-hot coals in one hand and a packet of sulphur in the other; the vessel was placed on the floor, and the fumes were given off. The operator remained in the building as long as he could stand it. This was regarded as the limit of safety for the animals. He then came out, bringing the burning sulphur with him; the door was closed, and the animals left to inhale the fumes. This job was frequently given to me to do

because it was said I could stand the sulphur fumes longer than most people, but I have an idea that it fell to my lot because I was young.

Another treatment was the administration of medicine by the mouth, turpentine and oil being a favourite remedy, but both these methods have now been discarded in favour of direct application into the windpipe of the agent to destroy the parasite by means of the ordinary hypodermic syringe or a special syringe for the purpose. It has been found that this method of treatment has been attended with a greater amount of success, but this naturally depends upon the extent to which the lungs have become involved before treatment is commenced; for it follows that in those portions of the lungs that have become consolidated no circulation of air is taking place, and the vapour given off by the drug will not to any extent reach such portions; further, that the fluid injected follows the laws of gravity, and runs from the windpipe into the first and lowest bronchial tubes to which it comes, which are those situated in the front portions of the lungs. Despite these drawbacks, this is the most successful line of treatment at present known.

Amongst the tried intratracheal injections which have been found successful are:

Turpentine	15 to 30 drops.
Chloroform	8 to 15 ,,
Carbolic acid	5 to 10 ,,
Olive oil	1 to 1½ drams.

Mix. The small dose for lambs and young pigs, the larger for calves.

Another is:

Black poppy oil	100 parts.
Oil of turpentine	100 ,,
Carbolic acid (two)	2 ,,
Purified cade oil	2 ,,

Mix. From 1 to 2 drams should be injected.

The injection should be made slowly and repeated daily for three days at least. But intratracheal injections would be somewhat difficult to carry out in the case of young pigs, and it would be a very onerous task to treat a large flock of young sheep and lambs. It is easiest and most successfully carried out in the case of calves.

The above are known as oily mixtures, and whilst they possess

the advantage of remaining longer in the air passages than watery solutions without being absorbed, the drugs dissolved in oil act less rapidly and energetically.

The following watery injections are recommended:

1. A 1 per cent. carbolic acid solution, the dose being 5 c.c. for lambs and 20 c.c. for calves, to be injected once a day for several days.

2. A 0·1 to 2 per cent. solution of potass. picronitrat., the dose being 5 c.c. for sheep and 20 to 60 c.c. for calves, according to age.

But whatever system of treatment is adopted, to be successful it requires to be supplemented by good nourishing food. The general health and condition of the young animal must be supported and maintained until it arrives at that stage when it would appear that the system is able to overcome the effects of these worms and to cast them out.

VERMINOUS BRONCHITIS IN EQUINES

The worm causing this disease, which is said to be but seldom seen, is known as the *Strongylus arnfieldi*. It is a white filiform worm, the male being from 1 to 1½ inches in length and the female about 2 inches.

In 1917 the writer observed a worm answering the above description, with the exception that many of the specimens were quite 3 inches long, in the windpipe and bronchial tubes of donkey foals and young donkeys in Bulawayo. There were about one hundred colts and foals running with their mothers. Practically all the young animals, and many of the mares, showed marked symptoms of being infested with intestinal worms. No distinguishing ante-mortem bronchial symptoms were exhibited, but on making a post-mortem examination of two of the young animals both were found to be harbouring a number of these worms in the larger and smaller bronchial tubes. When a piece of lung was cut off and pressed in the hand, amongst the froth exuding from the small tubes would be seen two or three of these white delicate worms wriggling out.

The intestinal infestation of worms was particularly intense.

Other species of strongyles are:

Strongylus rufescens.—A filiform, reddish-brown worm, the male being slightly under 1 inch in length and the female about 1 to 1½ inches long, found in the sheep and goat.

Strongylus commutatus.—Male about 1 inch, female about 1½ inches long, found in the domesticated rabbit.

Strongylus pusillus.—Found in the cat.

Strongylus bronchialis canis.—But little is known of this worm.

These four strongyles, so far as I am aware, have not been observed as occurring in South Africa.

STRONGYLOSIS OF BLOODVESSELS

Strongylus vasorum.—In the adult stage this worm lives in the right side of the heart and the larger divisions of the pulmonary artery. The eggs laid by the female are arrested by the finer arterioles in the lungs, and these pass through all their phases of development, giving rise to a form of pulmonary strongylosis in the dog. This worm has not been observed in South Africa.

Sclerostomum equinum or armatus.—This worm, known as the armed sclerostome, the armed strongyle, and palisade worm, is the best known of the sclerostomes, and perhaps, next to *Ascaris megalocephala*, is the most frequently met with worm in horses in South Africa.

It inhabits the larger bowels, particularly the blind gut (cæcum), where it may be found in great numbers. Some of the worms are so very firmly attached by their mouth parts to the mucous membrane of the intestine that when one attempts to remove their hold it takes quite an appreciable pull to do so. They are in colour grey, many of them distinctly reddish, about 1 inch to 1½ inches long, the female being the longer of the two. The male is blunt at both extremities; the female is very similar, but tapers abruptly into a short tail.

Where these worms have been attached to the mucous membrane will be seen small dark prominences about a pin's head in size; these apparently tumify and get larger when a young worm has entered them and coiled up in the interior.

These worms in their young state, before having reached sexual maturity, have a great tendency to migrate by the bloodstream to different parts of the body, and they have at different times been found practically in all parts and all organs of the body. While in the adult stage they frequently establish themselves in the walls of arteries, causing aneurisms, and when lodged there may give rise to what is described by some authorities as embolic colic, and in some cases the rupture of these aneurisms is attended by fatal results.

Sclerostomum tetracanthum, also known as *Strongylus tetracanthus*, is a small worm of about ½ inch in length, which

frequently accompanies the former worm; it also inhabits the large bowel of the horse, and is usually found free in the bowel amongst the food. They are at times present in enormous numbers. Although they apparently have no tendency to migrate and wander outside the bowels like the foregoing species, they encyst themselves in the mucous membrane and are found in tumours.

Some observers consider these two species of worms as practically inoffensive. I have met with them many times in animals that have died from colic and in animals that have died from poverty, and I cannot help regarding their presence as most detrimental to their host, and as being the cause either directly or indirectly of death in many instances. I regard them as particularly harmful to young horses, mules, and donkeys.

Sclerostomum hypostomum.—Is a small white worm, ½ to 1 inch in length, that is found in the large intestines of sheep and goats, where it causes similar pin-pricks of the mucous membranes to those seen in horses.

Œsophagostomum columbianum.—This is probably the best-known worm of this family in South Africa, where it is fairly widely distributed, causing in its larval stage a condition known as nodular disease of the intestines of sheep, or *knopziekte.*

Dr. Cooper Curtice, United States Department of Agriculture, was the first to describe these nodules, and to show that their originating cause is a worm, which he named as above. He says:

"The life history of this parasite is known to us, except that period when the eggs escape from the intestine with the excrement to the time when the embryo is found in these intestinal tumours, which must remain in obscurity. After the embryo has returned to the intestinal canal, it makes its way through the mucous coat of the intestine and becomes encysted there. Their presence causes inflammation, and these caseous (cheesy) tumours are the result. Having completed their encysted stage of development, they break from the tumours, and begin their life in the intestines, where they complete their growth, and, becoming sexually perfect, produce eggs, which eventually go through the same cycle. The small white worms which are found in the large intestines, principally the colon, are the sexually mature worms referred to."

In ordinary cases the cheesy nodules produced by the encysted embryos are confined to the large intestines principally, and when such is the case they do not appear to interfere seriously with the health and condition of the sheep. But there are a large number

of cases in which these nodules are so numerous that there is hardly an inch of sound gut to be found from where the small bowel leaves the stomach to where the bowel ends at the anus. The whole of the intestines are in a state of chronic inflammation.

The adult worm that is found in the large intestine is from ½ to ¾ inch in length; the larvæ that are found in the nodules are only about ⅛ inch in length.

Treatment.—This is the same as in most other verminous infections; apparently it should be on preventive lines to afford the greatest amount of relief, but until the missing link in the life cycle of the worm is known, the destruction of the embryos in the intestinal tract of their host before they encyst would appear to be the correct treatment to follow with our present knowledge, and it may be presumed that the best way to effect this destruction is by keeping a vermicide lick continuously before the sheep. Other precautionary measures indicated are the avoidance of damp pastures and the drinking of stagnant water.

Œsophagostomum radiatum.—Jowett has observed a nodular disease in the intestines of cattle in the Cape Province similar to the disease in sheep, and he was of the opinion that it was caused either by the embryonic stage of the above worm or by the *Œ. inflatum*, but favoured the idea of the former worm being the cause.

Other worms of this family are:

Œsophagostomum dentatum.—A white or greyish-brown worm, ⅓ to ¾ inch long, found in the large intestines of the pig.

Œsophagostomum inflatum.—This is ½ to 1 inch in length, found in large intestines of cattle.

Œsophagostomum venulosum.—This is ¾ to 1 inch in length, found in the goat.

Syngamus.

Two worms of this family have been determined which infect the air passages of domestic poultry and other birds—*S. trachealis* and *S. bronchialis*. These parasites give rise to the disease known as "gapes" in poultry.

The first-named worm is the best known, and is commonly recognised as the red worm or forked worm, and occurs in Great Britain as an epizootic, particularly in pheasants. It is from ¼ to 1 inch in length, the copulation between the male and female is permanent, and the worms are so intimately connected when in this state that they present the appearance of a single

worm with two heads (forked). The worms do not separate during life, and it has been held that the eggs cannot escape from the female until her body becomes disintegrated. The worms are so firmly attached to the mucous membrane of the trachea that they cannot be pulled therefrom without tearing them. The eggs and dead worms are coughed up by the infected birds, and the infection is held by some observers to be direct by other birds readily eating this sputum; others are of the opinion that a stage of the life cycle may be spent in an intermediary host, such as the earthworm.

Gapes is present in poultry in South Africa, but probably not to the extent that it is in Europe.

Treatment.—At one time a treatment of introducing a feather soaked in turpentine into the windpipe, turning it round, and withdrawing it with a certain number of the worms attached was advised; this required some manipulation, at times was attended with fatal results, and on the whole was unsatisfactory. The latest treatment for which satisfactory results are claimed is the intratracheal injection of 1 c.c. of a 5 per cent. solution of salicylate of sodium. The parasites are said to drop off after the injection, and are expelled by violent attacks of coughing.

Anchylostomum (Hook-Worms).

The best-known species of this family is the *A. duodenale* in man, causing a disease known as Egyptian chlorosis, intertropical anæmia (America), and miner's anæmia.

A. trigonocephalum is a small hook-worm about ½ to ¾ inch in length, found in the small intestines of dogs and foxes, causing a grave anæmia, and inducing as one of the principal symptoms an intermittent bleeding from the nose, accompanied by a swelling of the limbs and dysentery.

The disease has most frequently been seen in packs of hounds, infection taking place by the eggs of the worms being dropped into the drinking water by the infected animals. The disease has also been seen in cats, causing a similar anæmia.

Treatment.—Consists in cleanliness, worm treatment, and nourishing food.

Globocephalus is represented in the domestic animals by one species in the intestines of the pig.

Stephanurus is represented by one species (*dentatus*), which has been observed in America in the fat around the kidneys in pigs.

Ollulanus is represented by one species (*tricuspis*), a worm about $\frac{1}{25}$ inch in length, found in the stomach of the cat.

Hystrichis are filiform worms found between the muscular and mucous coats of the digestive canal of birds.

Physaloptera are found in fowls in Brazil.

Trichotrachelidæ.

The trichotrachelidæ comprises the following genera: Trichocephalus, Trichosomum, and Trichina.

Trichocephalus.—The front portion of the body is very thin, the posterior portion is thick and round in the males, the tail (thick part of body) is coiled up. There are several species found in ruminants, pig, dog, and rabbit.

Trichocephalus affinis.—Is so called because of its resemblance to the trichocephalus of man. The worms are from $2\frac{1}{2}$ to $3\frac{1}{2}$ inches long, and inhabit the large intestine, particularly the blind gut (cæcum), and are found in the sheep, goat, and ox. It attaches itself very firmly to the mucous membrane, but causes no trouble. The worm does not require an intermediary host in its development.

Trichocephalus dispar is found in man.

Trichocephalus crenatus is found in the pig.

Trichocephalus depressiusculus is found in the dog.

Trichocephalus unguiculatus.

These worms bear a great resemblance to *T. affinis*.

Trichosomum.—The trichosomes greatly resemble the foregoing genus. There are several species of these worms, which principally inhabit the members of the feathered world.

Trichina.—The genus Trichina comprises only one well-authenticated species—the *Trichina spiralis*.

TRICHINOSIS

Trichinosis is a disease caused by the presence of the *Trichina spiralis* in various animals, particularly in man and the pig.

There are two forms of the disease—muscular trichinosis, which is the most important, and is caused by the larvæ of the worms in the muscles of the body; the other form, which precedes it, is intestinal trichinosis, caused by eating trichinosed flesh; the larval worms in such flesh develop in the intestines and propagate their species.

The adult worms are only found in the intestinal canal, and,

being only from $\frac{1}{20}$ to $\frac{1}{6}$ inch in length, are scarcely visible to the naked eye. These worms are also very slender and tapering in the front half of the body. They are ovoviviparous, the embryo developing inside the egg whilst the latter is still within its mother's body.

The larvæ are found coiled up in a spiral or **S** form in cysts amongst the muscle fibre. They are less than $\frac{1}{25}$ inch in length.

History.—Peacock, in 1828, was the first to observe this disease, and deposited a fragment of muscle in Guy's Hospital, London, where it is still to be seen. In 1859, inspired by the alarm caused by the frequency of trichinosis in Germany, Virchow gave a dog some portions of human muscle invaded by trichinæ, and three and a half days afterwards found some worms in the intestines that he thought were adult trichinæ; but early the following year Professor Zenker of Dresden found a number of larval trichinæ in the muscles and adult worms in the intestines of a young woman who was supposed to have died of typhoid. On enquiring into the history of the case, he discovered that this young woman, some days before she was taken ill, had eaten raw pork, and, further, that other people who had partaken of the same pork had developed morbid symptoms; and on examination this pork was found to contain encysted trichinæ similar to those in the human muscles.

Zenker's observations, and the experimental investigations that followed them, may be said to have laid the foundations to the knowledge of this disease. The disease has been observed in every European country, but it is more prevalent in Germany, Russia, Holland, Sweden, and Denmark, and still more so in the United States of America.

Nature.—Trichinosis, in whatever animal it may occur, owes its origin to the eating of trichinosed flesh or excreta; therefore it follows that the disease is commonly found in flesh-eaters, but this applies to the mammals principally. Birds eat the flesh, and the worms develop to a greater or less extent in their intestines; but the larvæ do not as a rule become encysted in their muscles.

Man contracts muscular trichinosis by eating the flesh of pigs, and pigs contract the disease by eating the bodies of their dead companions or of mice and rats, which are very susceptible to the disease, or by eating excreta in which are mature worms or their embryos. Rats and mice contract the disease in a similar manner. The disease has been seen in a great many animals,

and a still further number have been experimentally infected with it.

When trichinosed flesh is ingested, the cysts enveloping the larval worm are dissolved by the gastric juice, and the larvæ are set free in less than twenty-four hours, and these before the fifth day have arrived at the adult stage. It has been estimated that a single female may give rise to 10,000 to 15,000 embryos; these are consequently in crowds in the intestines, and how they get from there into the muscles in the different parts of the body is not actually known; but it is surmised that they pass through the intestinal wall, and by utilising either the blood or the lymph-stream, reach their destination, which it is held may be as early as the sixth day after ingestion of the trichinosed meat. The adult worms are found in the intestines, particularly in the small intestine.

The encysted trichinæ possess remarkable vitality; they are said to resist for 100 days the putrefaction of flesh in which they are encysted, and they have been found alive in a ham that was in brine for twelve days, then smoked, nine months afterwards. They are destroyed by heat, such as boiling and roasting, if the meat is thoroughly cooked quite through the joint; freezing for some weeks also kills, but the amount of heat or cold and the length of time it must be applied to bring about the death of all the cysts are disputed points. The same remarks apply to salting and smoking.

Symptoms.—The disease is more frequently met with in the pig than any other of the domesticated animals, but it is the exception for these animals to manifest any symptoms unless the infestation is extraordinary. In such cases intestinal trichinosis will develop from the third to the tenth day after infection (eating the flesh). The animal loses its appetite, is very thirsty, grinds the teeth, is very dull and feverish, back arched, belly tucked up, painful on pressure, and diarrhœa is present. These symptoms continue for about a month.

From the eighth to the fifteenth day the symptoms of muscular trichinosis commence: the limbs are stiff, the movements are halting and uncertain, the hind-quarters may be more or less paralysed, the joints and muscles are painful.

These symptoms are present, but intensified, in man, and are accompanied by vomiting, perspiration, swelling of the muscles of the face, etc.

Treatment.—There is no known curative treatment, and the preventive treatment consists in keeping pigs under such con-

ditions that they cannot obtain trichinosed flesh or excreta, and in this connection the fact of rats and mice being subject to this disease must not be overlooked.

Filariidæ.

The body of these worms is long and filiform, and the tail of the male is usually rolled up. They are divided into four genera: Filaria, Spiroptera, Dispharagus, and Tropisurus. The first two alone furnish intestinal parasites. The two others are met with in the gullet and stomach of birds.

Filaria.—The Filariæ are more particularly parasites of the serous membranes and the subcutaneous connective tissue. They belong to the worms that are very rarely met with in the intestines of the horse and the dog.

FILARIA OF THE SKIN

Filaria hæmorrhagica.—The expression "sweating blood" is a fairly common one, but it is doubtful if many of the persons using it know it is actually a condition that occurs in the horse, and is caused by the above worm.

It appears from researches that the Chinese were aware from the remotest antiquity of a breed of horses in Khodang which perspired blood. The disease is special to horses of Asiatic origin, but has also frequently been seen in Hungarian horses; it has occurred in France and in Spain. It has been observed in England amongst imported horses, and cases have been seen of it in South Africa in Hungarian horses.

Symptoms.—The disease is characterised by hard, painless elevations of the size of a pea to that of a hazel-nut, particularly in the shoulders, the sides of the body, neck and withers, and on the back. These elevations are formed by an accumulation of blood, and they rupture in an hour or two after their appearance, or it might perhaps be better described as the blood oozing out similar to sweat, as the openings through which it comes are scarcely visible, and these usually disappear and nothing further is seen at the particular spot, but the eruption goes on for several days at other places. If it is desired to find the worm, the elevation must be opened up before the blood has commenced to ooze, when the worm will be perceived, but it contracts to bury itself in the connective tissue.

The worm causing the disease is white and filiform, about

2½ inches long, and it is ovoviviparous. Its life history is unknown.

The disease is usually not attended with any serious consequences, but with considerable inconvenience by the elevations of the skin coming in contact with the saddle and harness.

Summer Sores of Horses.

This is a disease in which the ordinary and other wounds on horses, such as those due to the harness, assume an intractable character and do not heal up for some months. These wounds have a tendency to spread and become soft and pulpy, with reddish-brown granulations; they cause the affected animal intense irritation, consequently the surface of the wound is frequently enlarged by the animal gnawing or rubbing it.

The disease is met with in hot countries, and has been more frequently observed in Italy and Algeria than elsewhere, particularly in the donkey.

The worm causing it is only about ⅛ inch in length, and is regarded as a larval form, and has been named the *Filaria irritans*.

SUBCUTANEOUS FILARIA

Filaria medinensis (*Guinea Worm*).—This worm is chiefly known as a parasite of man, but it has been found in other animals—the dog, horse, and perhaps the ox. It is peculiar to hot countries—Arabia, India, and in Africa; it is found in Guinea, Upper Senegal, East Africa, and Egypt. It was introduced into South America by negroes at the time of the treaty, and has become endemic in Guiana and parts of Brazil.

The worm is found in the subcutaneous connective tissue, and in man is commonly met with in the legs and feet. It is usually rolled up in a spiral manner, and causes the formation of superficial tumours. In the dog these tumours are oblong and flattened, and feel like a skein of thick twisted cord. At first they are painless, but they gradually become painful, until eventually the poor brute cannot have any peace, but is constantly licking the tumour and restless. Finally, the tumour bursts in one or more places, one of which openings gives exit to the worm, which is intensely painful. The dog furiously licks the wound, and frequently seizes the worm in its teeth in an attempt to draw it out; but this is very painful, and the animal desists.

In man, rupture of the worm during extraction is a very grave,

often a fatal, accident; in the dog this does not appear to be so.

The number of worms found in the same individual varies from one to five.

The usual method employed to extract the worm is to roll it round a bit of stick and to gradually keep rolling it up; it may take several days to get it away. Another method is to lay the skin open the whole length of the tumour.

It is a white worm from 2 to 12 feet long, and about as thick as a sewing-needle. Only the female worm is known, and the body is engorged with eggs and embryos that can only escape by the rupture of the mother worm.

FILARIA OF THE BLOOD

Filaria immitis is a thin, white, round worm, ovoviviparous. The male is from 5 to 7 inches long, and the female is from 10 to 12 inches in length.

It chiefly inhabits the right heart and pulmonary artery in the dog. It has been met with in various places on the continent of Europe, and is very frequent in Japan and China. In this latter country it kills nearly all the European dogs.

Death usually results from this form of verminous infection. If the worms are present in large numbers, they become interlaced in bundles that obstruct the circulation. When worms are present, a microscopical examination of the peripheral blood will usually reveal the presence of eggs and embryos of this worm. It is supposed that infection takes place through drinking water, as is generally admitted it does with regard to *Filaria sanguinis hominis*. The culex mosquito, when it sucks the blood of people who are harbouring this worm, takes in with it the embryos, which develop in its digestive tube, and in about five days they measure about $\frac{1}{25}$ inch. At this period the mosquitoes resort to stagnant water to lay their eggs and die, and the worms escape and live at liberty in the water. It is in drinking this water that man becomes infected.

This verminous infestation of the blood in man is found in the hot parts of Asia and America.

Filaria sanguinis equi.—Larval forms of a worm were found in a horse in Egypt to which the above name was given.

Filaria evansi were similarly found in a camel.

Filaria recondita.—Only the embryos of this worm are known (hæmatozoa of Lewis), and are found in the blood of dogs, particularly in China and India.

FILARIA OF THE SEROUS MEMBRANES

Filaria equina or papillosa is a white filiform worm, ovoviviparous, the male being about 2 to 3 inches long, and the female 4 to 6 inches in length. This worm is frequently met with in the abdominal (peritoneal) cavity, and more rarely in the chest (pleural) cavity in the horse, donkey, and mule, particularly in animals in poor condition. It has also been found in other parts of the body and in the scrotal sac. I have come across this worm, or, to be accurate, what I have taken to be this worm, on more than one occasion when castrating donkeys in Rhodesia.

FILARIA OF THE OX

Filaria cervina or Labiato papillosa or terebra.—This worm much resembles that of the horse, and is found in similar situations.

FILARIA OF THE EYE

Filaria pellucida.—This name has been given to a worm found within the eyeball of the horse, but it is doubtful if it is not a young form of *Filaria papillosa.*

The disease is frequent in horses in India, and has been observed rarely in Europe and America.

In the ox a similar infection of the eyeball has been observed which is thought may be due to larval forms of *Filaria cervina.*

Worms outside the Eyeball.—Filariæ have been seen several times beneath the eyelids of the horse and ox, and even in the lachrymal canal of the horse. They often cause conjunctivitis.

Filaria palpebralis is a small white worm from $\frac{1}{3}$ to $\frac{2}{3}$ inch in length, found beneath the eyelids in the horse.

Filaria lachrymalis is a similar but a longer worm, the largest specimen being an inch in length.

The presence of these worms causes inflammation of the mucous membrane (conjunctivitis) covering the eyeball and eyelids. This may lead to ulceration of the eyeball, and result in the loss of the eye. The disease is frequently met with in South Africa.

Treatment.—This consists in removing the worms. Secure the animal, and then with a feather clean out the space between the eyeball and the lower eyelid and the haw of the eye; if worms are present, they will be caught on the feather. One of the best dressings to apply is tobacco water or a solution of corrosive

sublimate 1 in 1,000 of water. If the eye has become ulcerated, boracic acid may be dusted into the eye, or a saturated solution of same may be applied as a lotion.

FILARIA OF THE INTESTINES: POULTRY

Filaria gallinarum.—This worm was investigated and described by Theiler in 1918, who gives the length of the worms as —male about 1½ inches, female about 3 inches.

His attention was called to these worms some years ago by a farmer in the Orange Free State, who discovered that when certain large white ants are dropped into hot water, in some of them the bodies burst and the exit of the worm takes place. This ant exists widely in South Africa, and is known by different names in different parts. Specimens submitted to Claude Fuller were found by him to belong to *Hodotermes pretoriensis* (Fuller). This ant is greedily eaten by poultry. The life history of the worm was worked out by Theiler, with the exception that he was unable to determine whether it has three or four larval stages. It has but two hosts. The ants pick up the eggs passed in the fowls' droppings; the embryos hatch out, and undergo one change within the ant; the fowl eats the ant, and the other stages take place in its intestinal tract.

The ants that are the intermediary hosts of this larval worm have their abdomen blown up more like a balloon than is the case with those uninfested. It certainly looks most strange to see a worm 1 inch long wriggling out of the abdomen of this comparatively small ant, as I did in Sir Arnold Theiler's laboratory.

SPIROPTERA.—These worms are very similar to the Filaria. The body is not so long, but they differ but little except in the places in which they are found.

Spiroptera megastoma.—These are small worms, ¼ to ½ inch in length, that are found in tumours usually situated in the right sac of the stomach of the horse. These tumours are frequently about the size of a Barcelona nut, are perforated, and on pressure greyish matter and these worms are extruded.

Spiroptera microstoma.—These worms, similar to the foregoing, but slightly longer, have been found in great numbers in the stomachs of horses and donkeys in France. The head is usually engaged in the mucous membrane.

Spiroptera strongylina.—About the same size as the foregoing two, and has been met with in the stomachs of pigs in Germany.

Spiroptera sanguinolenta.—This worm is blood red in colour, male 1 to 2 inches, female 2 to 3 inches in length, and is found in tumours of the stomach and gullet of the dog, fox, and wolf, particularly in China and Brazil, also in Italy and France. The worm finds its intermediary host in the Eastern cockroach (*Periplaneta orientalis* Linn.).

Spiroptera Reticulata.—This worm is found frequently in the flexor tendons of the foot of the horse in Kazan, and causes lameness and the formation of tumours. The female worms appear to measure 15 to 20 inches, but as they are rolled round muscular and tendinous fibres it is impossible to remove them intact.

Gnathostomidæ.

This family is established by the only genus, Gnathostomum. The gnathostomes are parasites of the stomach, either of the pig or of the dog and wild cat.

Gnathostomum hispidum.—This worm was found by a Russian traveller in the stomach of the pig. It has been seen in Vienna, and is known to the pork-butchers there as the tricoloured worm. The male worm is about 1 inch in length, and the female $1\frac{1}{2}$ inches long. The anterior extremity is provided with twelve rows of chitinous plates, the summits of which are garnished with sharp hooks directed backwards. By means of these it fixes itself to the mucous membrane of the stomach, and lives on the blood it extracts.

Angiostomidæ.

This family—not well characterised—comprises the genus Strongyloides, of which the species live as parasites in the intestine of man, the sheep, pig, and rabbit.

Strongyloides.—The best known of this genus is the species parasitic in man causing a pernicious diarrhœa, often known as Cochin-China diarrhœa. The worm causing this disease is known as *Strongyloides intestinalis*, and was formerly thought to be caused by two worms known as *Anguillula intestinalis*, a worm the female of which alone is known, about $\frac{1}{12}$ inch in length, and found in the small intestines of man; the other worm is known as *Anguillula stercoralis*, and is found in the fæces of persons suffering from this disease, and is barely $\frac{1}{25}$ inch in length. This latter worm is now recognised as merely the free state of the first.

PART III

DISEASES CAUSED BY AN ULTRA-VISIBLE VIRUS

DISEASES IN WHICH THE VIRUS OR CAUSE IS INVISIBLE BY THE HIGHEST POWER OF THE MICROSCOPE

FREQUENTLY these diseases are grouped as "diseases caused by invisible bacteria." This is assuming, what is not certain, that the disease is due to a bacterium; it is possible that in some cases the disease is due to a protozoa.

The justification for asserting that a virus is not visible lies in the power of demonstrating that materials such as blood which are known to be infective, and must therefore contain the virus, are still infective after passing—certain precautions being taken —through an efficient earthenware filter. One is entitled to say that such a virus is ultra-visible.

The proof that there existed ultra-visible infective viruses was proved by Beijerinck in 1898 in a tobacco-plant disease, where necrotic spots appear on the leaf which are contagious. He found that infusions of the leaves after passing through an earthenware filter were still infective. He did not conclude that the virus was an invisible bacterium, but that it was an entirely new thing, which he called the *Contagium vivium fluidium*.

The advantage of grouping these diseases together lies in the ease in which they can be compared one with the other, and the similarity in some cases between them.

RINDERPEST

Synonyms.—Cattle Plague (French, *La peste bovine;* Italian, *Peste bovilla*).

History.—The original home of rinderpest was in Asia. It is supposed to have been brought from China by the Huns to the basin of the Caspian Sea; it was carried thence by them and others to Western Europe, and by the migration of peoples westward it was transported over the entire continent, where it intermittently raged from that time until nearly 1880. Extensive outbreaks which appeared were usually associated with great wars, the cattle following the troops disseminating the disease over distant territories. In this respect the wars of Charlemagne in the ninth century, and the incursion of the Mongols into Europe in the thirteenth century, are noted for widespreading the disease.

In 1744, it is said, the disease was first introduced into England from the continent of Europe. In 1865 an extensive outbreak of the disease occurred in Great Britain, which originated in a cargo of cattle sent from Revel to Hull. It was stamped out after eighteen months, but caused enormous losses. Another outbreak occurred in 1872, due to cattle imported from Russia, but it was quickly stamped out. The last outbreak of the disease in Great Britain occurred in 1877.

In 1841 it was introduced into Egypt by the importation of animals from Austria. It was imported into Abyssinia in 1890 by cattle supplied for the Italian Army.

Sir John Kirk, writing to *Nature*, states it was in Somaliland in 1889. Captain Lugard refers to its presence in Masailand towards the end of 1890. Sharp mentions it as being prevalent to the north of Nyassaland in 1892. In 1893 it was on the shores of Lake Nyassa. It is not a great many years back when Central Africa was "Darkest Africa," and little was known of what happened there. Probably the first news of the approach of rinderpest was that obtainable in Bulawayo in the early months of 1896, when news came to hand of its existence north of the Zambesi River, and people had not long to wait for further con-

firmation of its existence. It is believed to have been brought to the south bank of that river by some missionary cattle that travelled down through the Mafungabusi district. The next report was that it was killing a great number of game in the Inyorka district. It was not at that time recognised as rinderpest, but was called "Zambesi cattle fever" and "black gall-sickness." Gray identified the disease and reported upon it early in 1896, and by March of that year it was at Bulawayo, from whence it spread far and wide, leaving a never-to-be-forgotten stinking desolation behind it. The country was full of cattle and big game, both of which the disease decimated; it destroyed about 97 per cent. of the cattle. Khama estimated his losses in Bechuanaland at one million head, and as the Matabele were richer in cattle than Khama, it is probable the losses in Matabeleland were about a million and a half. The only means of communication between Rhodesia and the south was by road, and all along this were carcases of rotting dead animals; in consequence, one unending stench stretched from Bulawayo to Palapye.

Naturally, once the disease had invaded Rhodesia, the news of its nature and the rapidity of its spread caused great alarm in the southern colonies, and the deaths from the disease of 20,000 cattle at Vryburg, *en route* from Bulawayo to Cape Town, further increased the consternation. Various attempts to check its progress south were made, including the erection of fencing, but to little purpose. In 1897 the Transvaal lost 980,000, and the Cape Colony losses in the years 1897 and 1898 were estimated at 1,300,000 cattle. At the invitation of the Cape Colony, Professor Robert Koch came to South Africa, and by his investigations, coupled with those of other investigators, including Danysz, Bordet, Theiler, and Pitchford, methods were found of combating the disease which have since proved of inestimable value to other countries, as they did at that time to South Africa. Rhodesia, having had the full shock of the disease and being practically denuded of cattle, was the first country in South Africa to be free of it; Basutoland and the Orange River Colony were free of the disease in 1902; Swaziland, Portuguese East Africa, and the Transvaal were clear in 1903; and at the end of that year the only places in South Africa in which it was present were German West Africa and Zululand.

After such an experience naturally South Africa has always kept a watchful eye on Central Africa and the north. In 1913 the writer was despatched, on behalf of the Rhodesian and

Union Governments, to German East Africa, to report upon the condition and nature of rinderpest as it existed in that country, and the probability of its extension south. In September, 1915, Mr. G. C. Hooper Sharpe, M.R.C.V.S., also of the Rhodesia Veterinary Service, was similarly sent up to the border of Northern Rhodesia and German East Africa, to remain with the troops guarding that boundary and watch for evidence of the disease travelling south. Later on he was followed by Mr. C. E. Gray, Principal Veterinary Officer of the Union, who took charge of, and successfully directed, the measures to eradicate the disease in the southern portion of German East Africa, and prevent its spread southward. All the territories in South Africa shared in the steps taken, and those who could sent veterinary surgeons, rinderpest serum, etc.

In 1918 some anxiety was caused by the report that the disease had broken out amongst the cattle accompanying Von Lettow Vorbeck, the leader of the German East African troops, when he crossed over into Northern Rhodesia and surrendered; but prompt action, including the destruction of all the cattle, was taken, and no further evidence of the disease south of German East Africa has since been manifested.

The disease has never appeared in America, Australia, and New Zealand. The position at present is that the disease is endemic in a great part of Asia, including India, Asia Minor, and Turkey; it is also endemic in Abyssinia. In 1913 it was undoubtedly endemic in the herds of nomadic natives, and probably during the war it will be found to have become endemic over considerable portions of North-East Central Africa. In this form it does not destroy a great percentage of cattle, and few, if any, game; but there is the possibility of even the latter contracting the disease, if only in a mild form, and of transmitting it to susceptible animals. In this and the recognised tendency of the disease of assuming at times a virulent and epizootic character lies the danger to South Africa.

Nature.—Rinderpest is an acute, febrile, contagious, infectious disease of cattle that occasionally passes to other ruminants, and is caused by an ultra-visible virus.

The disease is met with in two forms, either as an enzootic or as an epizootic. Perhaps an intermediary form between these two should also be mentioned, in this sense: In those herds in which the disease is enzootic but a small percentage of deaths occurs; bordering on these herds there are herds that have had the disease sundry times, but in which the outbreaks are inter-

FIG. 31.—RINDERPEST IN BECHUANALAND PROTECTORATE: CATTLE SHOT AND DEAD FROM THE DISEASE. (Photograph by J. S. Loosley.)

In one small valley 5,000 cattle were said to be lying similar to the above.

mittent and a larger percentage of deaths occur in this intermediary zone; outside this, and farther away from the endemic areas, the more susceptible the cattle become, consequently the more virulent the disease and heavier the mortality.

It was noted in German East Africa in 1913 that in some of the herds infected only young stock—that is, calves and animals up to three and a half years old—died, whilst in other herds animals of all ages died.

Although in most of the instances recorded, when the disease has spread over Europe and Africa as an epizootic, these incursions have been due to the movement of cattle, it appears as if it is the nature of the disease to lie in its endemic centres in a more or less passive state for a number of years, and then, through undetermined reasons, it assumes a more virulent form and an epizootic character.

It also appears as if the disease has a tendency after a time to lose some of its virulence in an epizootic outbreak; for instance, the percentage of losses was not so severe at the end of the disease in the Cape Colony as it was in its earlier stages in Rhodesia and Bechuanaland. The mortality from the disease varies from 30 or less per cent. up to nearly 100 per cent.

In the main it is a disease of cattle, but it may also cause considerable losses in flocks of sheep and goats, and in its epizootic form it is very destructive to wild ruminants. When it swept through Rhodesia in 1896 it practically denuded the country of buffalo, the larger antelope such as eland and koodoo, and wild pigs. Prior to its advent the country had immense herds of these animals, and large belts of country infested with tsetse fly that fed upon the game. When the disease was over, tsetse fly had disappeared from Southern Rhodesia, except from one or two small areas towards the Zambesi River. Other species, like sable, tsessebe, and roan antelope, were practically unaffected; these and the remnants of the game left or missed by the rinderpest wave again increased to considerable numbers, and the tsetse fly has likewise increased, and is extending its area within its old haunts.

The virus of the disease is present during the entire period of sickness in all tissue fluids.

In the blood, according to Theiler, the virus is apparently adherent to the red blood-corpuscles, and is not present in the serum unless it is blood-stained. Besides the tissue fluids, the virus is present in different secretions—saliva, nasal

discharge, urine, dung, tears, vaginal discharge, and perspiration.

The disease is spread under natural conditions by direct contact with infected animals, or is transmitted through the raw products of infected carcases such as meats, hides, etc., or by materials that have been contaminated by such carcases or by infected secretions and excretions. In most instances the disease is disseminated by the movement of infected animals. Animals only slightly infected may transmit the disease for a period of fourteen days, and chronic cases probably for a longer time; but food, drinking water, stable utensils, and clothes, and persons by their feet, may also transmit or transport the infection.

Infection usually occurs through the digestive tract. Popular belief, probably founded on the ready manner in which the disease spreads owing to the great susceptibility of cattle, is that the infection spreads by means of the air. This appears to be erroneous, for although it has not been absolutely proved that it is not so, the experiments that have been carried out indicate that it is improbable. The incubation period is from three to ten days.

In severe cases the disease lasts from four to seven days, in less severe ones fourteen or more days, and recovery in the cases that do recover extends over two or three weeks. In the milder and enzootic form of the disease the symptoms do not develop to such a degree, and may disappear after a few days of illness.

Camels are also susceptible to the disease, but horses, mules, donkeys, and carnivora are not, neither is man. Sheep and goats are susceptible, and natural outbreaks have occurred amongst them; but they are not very susceptible, and the virus is attenuated by passage through them.

Symptoms.—The first symptom is a rise in temperature to 104° or 105° F. This is present before the animal shows external signs of the disease. On the second or third day of the fever the animal is noticed to be weak, the coat staring, and the pulse increasing in frequency—over sixty beats to the minute.

After a few days of fever diagnostic symptoms of the disease appear, the first symptom usually being inflammation of the mucous membranes of the eyes, attended with swelling and partial closing of the eyelids, and accompanied by a watery discharge with a similar discharge from the nostrils. This discharge gets more profuse daily, and changes from a watery to a mucoid and then sticky, purulent character. At the same time a diphtheritic inflammation of the nasal mucous membrane is

taking place. This sometimes extends backwards to the throat; if so, a husky cough will be present.

This is followed by a diphtheritic inflammation of the mucous membrane of the mouth and lips, which usually begins on the lips near the incisor teeth. It spreads quickly, and is frequently found next on the inside of the cheeks and under the tongue; at the same time the process of erosive ulceration—that is, first inflammation, then encrustation, followed by desquamation—is commencing at several different places in the mouth. In some cases the mouth is just one large diphtheritic ulcer. Increase of salivation naturally follows these changes in the mouth.

At first the appetite is good, but with the advent of the mouth lesions it is lost, and rumination also diminishes or ceases entirely. The condition of the bowels in the early stages of the disease is changeable; one day the animal is constipated, the next day diarrhœa is present. This changeability is followed by an offensive diarrhœa, which at first is mixed with mucus, then with blood; finally it is passed involuntarily through a flabby and open anus.

In females an ulceration of the vagina attended with a mucoid purulent discharge is seen.

Pregnant females abort in the early stages of the disease, even though they are not more than two months pregnant.

As the visible symptoms increase in intensity the temperature usually drops in proportion, frequently falling below normal.

In the last stages of the disease the animal gets very weak, does not eat, is very thin, lies down and is unable to rise, the coat is staring, and the ears hanging. The discharge from the eyes, nose, and mouth is partly dried, and hanging in long strings. The part around the anus and the under surface of the top of the tail is covered with offensive smelling fæces mixed with mucus and blood.

The preceding is a description of the typical cases that were met with in German East Africa; there were many cases that were not typical, but in 90 per cent. the mouth symptoms and conjunctivitis (inflammation of the eyes) were present. In a few cases an eruption (breaking out) was seen on the skin. The under surface of the neck was the most frequent site for it to occur, but it was also seen on other parts of the body, particularly the back. It was not determined whether this occurred in uncomplicated cases of rinderpest, or whether it was due to a mixed infection of malignant catarrhal fever. This exanthema, when present, shows itself in the form of lenticular nodules, from which a serous fluid

exudes that mats the hair together, forming a crust, which either becomes desiccated, or if it is rubbed off it leaves a small ulcer. If many nodules are present by the exudations running together, big continuous crusts are formed.

It must also be remembered that the foregoing symptoms and their sequence of occurrence are related, as they were present in rinderpest in German East Africa, where the mortality ranged from 30 to 80 per cent. In more virulent outbreaks of the disease, where the mortality approximates 100 per cent., the symptoms would probably be presented more quickly, follow each other with greater rapidity, and result in death in a less space of time.

Post-mortem Appearances.—The body is very thin and poor; there are crusts of dried mucus and pus around the eyes, nose, and mouth, and dried fæces mixed with mucus and blood round the anus and tail. Sometimes exanthema is seen on the under surface of the neck and body; the mucous membrane of the eye is inflamed, and a mucoid purulent fluid in the cavity inside the eyelids.

The fluid in the heart sac is increased in quantity, and not clear in colour; the heart muscle is degenerated, and the heart filled with clotted blood, with blood-spots on its inside and sometimes on the outside also.

The mucous membrane of the nasal cavity is red and swollen, and containing ulcers of various sizes, usually elongated in shape. The mucous membrane of the throat is inflamed and containing blood-spots.

In the lungs there is usually nothing to be seen.

The fourth stomach is usually empty; it sometimes contains a chocolate-coloured fluid. The mucous membrane is swollen and cloudy, and on top of the folds it is hyperæmic. In some cases it is of a dark red colour throughout. Near the pylorus ulcers of various sizes are mostly found.

The small bowel (jejunum) contains thin liquid contents. The mucous membrane is swollen and hyperæmic, and in folds that cannot be pulled apart. Sometimes it is a very dark red colour throughout its length. Near the ileo-cæcal valve on Payer's patches ulcers are almost invariably found.

In the cæcum and colon the mucous membrane is swollen, the submucous tissue œdematous, and ulcers of different sizes are usually found.

The rectum is empty, as if it had been washed out; it shows stripes and hæmorrhagic patches.

The liver is swollen, yellowish-brown, or saffron-coloured,

friable, fatty when cut, and lobules not very distinct. In the bladder the mucous membrane is swollen and hyperæmic; in some cases hæmorrhagic. In the urine nothing is to be seen. In the vagina the mucous membrane is hyperæmic, sometimes ulcerated. The spleen is usually slightly enlarged, dark red in colour. When cut the trabeculæ are not very distinct; the pulp protrudes slightly.

The kidneys are grey-brown in colour, slightly flabby; the capsule is easily detached. .

Diagnosis.—In German East Africa at the time of the rinderpest another disease was present, malignant catarrhal fever, that has practically identical symptoms to rinderpest. This caused considerable confusion, and in some cases blood inoculation had to be resorted to, to determine which disease was present.

The following are the chief differences between these two diseases:

In rinderpest it is rare for the inflammation of the mucous membrane of the eye to extend to the eyeball; in malignant catarrhal fever it is the rule for it to do so, attended with opacity of the cornea—that is, the eyeball goes milky white and sometimes ruptures.

The exanthema, if it is ever present in uncomplicated rinderpest, is present in only a very small percentage of cases; in malignant catarrhal fever it is present in 90 per cent. of the cases.

The final distinguishing test between the two diseases is that malignant catarrhal fever is not conveyed by blood inoculation, and rinderpest is.

Treatment—Medicinal treatment has been found to be of no help to infected animals. In many countries attempt at such treatment is prohibited, as all animals suffering from rinderpest have to be destroyed. In other countries, including South Africa, treatment of the disease by means of inoculation is carried out; in fact, it was in South Africa that this method of treatment was perfected, and it has not only rendered immense services to South African agriculture, but to numerous other countries that have been ravaged by rinderpest.

When Professor Robert Koch came to South Africa to investigate the disease, upon testing the infectivity of various secretions his attention was drawn to certain properties possessed by the bile of animals which had died from rinderpest, the injection of which into healthy animals was found to confer a certain degree of immunity. The method possessed certain disadvantages. The bile had to be used within four days of its

collection; the animal had to be sick for six to eight days before its bile contained immunising properties. A beast dead of the disease only afforded sufficient bile to inoculate twenty to twenty-five animals, consequently it meant that a large number of animals had to be sacrificed to provide sufficient and satisfactory bile for inoculation. It did not confer immunity until the animal had been inoculated eight or ten days, during which time animals could become naturally infected. The immunity set up was of short duration, lasting not more than four to six months. All biles are not suitable for inoculation. Potent bile can be obtained only after the seventh day of the disease; it should be of a green colour, odourless, and on shaking should form a white foam; but if blood happens to become mixed with the bile, which it is liable to do when the bile is being collected, the injection of such material transmits the disease. Despite all these drawbacks, a great number of animals in South Africa were immunised by this method and saved to the country. The dose employed was 10 c.c. injected into the dewlap with a hypodermic syringe.

Edington improved on Koch's method by mixing 1 part glycerine with 2 parts bile, which enabled it to be kept for months and perhaps years. Large doses of the mixture could be injected with safety, the dose employed being from 15 to 35 c.c. injected into the dewlap; but it possessed only a temporary immunising power, and Edington advised later that it be followed ten days or a fortnight after its administration by a 1 c.c. dose of virulent blood. By this method a lasting immunity was conferred. The method in the hands of some inoculators proved fairly satisfactory, and few losses from inoculation occurred. With many others, however, the losses were heavy; it was necessary, therefore, to find some method of more general application.

Koch and others found that the blood-serum of animals that had recovered spontaneously from the disease possessed certain properties which would render resistant to infection susceptible animals into which it was injected. Bordet, Danzys, and Theiler devised a method of conferring immunity by the injection of immune blood-serum into cattle, which were afterwards exposed to natural infection, with the result that they contracted the disease in a mild form and became subsequently immune.

Kolle and Turner worked out a method at once simple and easily applied, which is known as the "simultaneous method," from the fact that a quantity of protective serum, the dose of which is determined by experiment, is injected simultaneously with a dose of virulent blood, usually 1 c.c., the serum and blood

being injected on different sides of the body. The serum is obtained from cattle that have been hyper-immunised—that is, repeatedly injected with virulent blood in increasing quantities. By this method the immunity conferred by the serum is immediate, but of short duration, and it is rendered active by the reaction induced by the virulent blood. Thus is conferred an immunity which is regarded as lasting and practically permanent.

Recovery from a naturally acquired attack of the disease conveys in the majority of cases a lasting immunity; in exceptional cases repeated attacks have been known to occur, but usually the infection was in a mild form.

The methods of inoculation now most frequently practised are those of the " serum alone " and the " simultaneous method."

In the " serum alone " method an immunity to the disease is conferred for from two to four weeks, according to the amount of serum injected. This method is employed to tide the animal over the time that it may become infected, and for dealing with isolated outbreaks that can be controlled and guarded against reinfection from outside sources.

The simultaneous method is employed in herds where active infection is present and in areas where the movements of cattle cannot be controlled. The immunity by this method is immediate and lasting. The objections to its general employment are the setting up of active rinderpest infection and the liability of conveying some other disease as well as rinderpest by the blood inoculation. In South Africa, where there are a number of diseases transmitted through the blood, the risk is a considerable one, that cannot be entirely overcome by previous microscopical examination of the blood; therefore it follows that the " serum alone " method, by which the risk of conveying other diseases is considerably lessened, is much preferred by many.

In case a farmer is in possession of salted cattle, and is so situated that he cannot obtain the highly immunising serum, he can utilise the blood of such animals. The salted animal may be either one which has recovered from a natural attack of the disease, or from an attack following inoculation. In any case, the animal selected should be in good condition, free from disease, and must have recovered from rinderpest for at least six weeks. It is better, where possible, to prepare or fortify these salted animals by injections of virulent blood, commencing with 10, then 20, 50, and lastly 100 c.c., at intervals of ten days.

Obtain a clean iron pail, enamelled if possible, or a tin pitcher with a closely fitting lid, wash it thoroughly, then fill with water,

and boil the water in it for a quarter of an hour. Make a whisk or brush of iron by unravelling a length of iron rope; boil this in the bucket or pail. Cast the salted animal, cut the hair of the neck from the jugular vein, wash it thoroughly, and then rub it with a disinfectant, such as 5 per cent. solution of carbolic acid in water, or sheep-dip and water. Empty the water out of the bucket or pitcher. Bleed the animal into it, and at the same time whip the blood with the iron brush until it is covered with the white fibrin of the blood. This will be accomplished in about ten minutes or a quarter of an hour. Strain this defibrinated blood through a well-boiled piece of muslin or butter-cloth. Inject this defibrinated blood in doses of 100 or 200 c.c. into the "sick" animals, under the skin only—not into the muscles—either behind the shoulder or into the dewlap.

INFLUENZA

Synonyms.—Catarrhal Fever, Infectious Catarrhal Fever of Horses, Horse Distemper, Epizootic Cellulitis, Pink-Eye, Epizootic Gastro-enteritis (French, *La grippe*).

History.—History indicates that influenza has been recognised in various outbreaks for several hundred years past. Even before the Christian era a severe epizootic, believed to be influenza, is recorded as having occurred in Sicily, and again in A.D. 301 it spread over a considerable portion of Italy, causing great loss amongst the war-horses of Rome. In 1648 the disease affected Germany and spread to other parts of Europe, and in 1711 it followed the tracks of the great armies all over Europe, causing immense losses amongst the horses. Other extensive outbreaks occurred at intervals from 1813 to 1883, one of which was that of 1870 to 1873 in connection with the Franco-Prussian War. One of the most remarkable and widely-spread outbreaks of influenza occurred in Canada and the United States of America in 1872 to 1873. It commenced in Toronto on October 1, 1872, and rapidly spread through Canada. It appeared in Buffalo, N.Y., on October 14; it extended to Ohio in the south, thence eastwards to the Atlantic coast and westward to California. In this epizootic the type of the disease was that known as "pink-eye." Various epizootics of the disease are recorded as having occurred in Great Britain from 1688 to the present time. The disease has occurred in South Africa at different times. In 1916 an outbreak of it occurred in and around Bulawayo, in which at least 75 per cent. of the horses, mules, and donkeys suffered, and the virulence of the disease affected these animals in the reverse order to which they are placed: donkeys were seriously ill, and several deaths occurred amongst them; in mules a few deaths occurred; but in horses the mortality did not probably exceed 1 per cent.

Nature.—It is an infectious febrile disease of the equine species, often occurring as an epizootic. Little doubt exists at the present day that its cause belongs to the ultra-visible virus group. It is characterised by a marked tendency to complications and

secondary infections, and by the variety of clinical types it assumes in different outbreaks. In consequence of this tendency it has become customary to recognise certain forms of the disease as catarrhal, pulmonary, gastro-intestinal, etc. The disease known and described as contagious pneumonia in horses is thought by many to be only and always a secondary infection of influenza, and whilst there are many others who doubt the correctness of this view, it is apparently gaining in favour. We in South Africa, after our recent experience of the Spanish influenza amongst human beings, and the frequency of the pneumonic complications in connection with it, will, I think, regard that view as probably the correct one; for although there is no evidence of influenza having been transmitted by man to the horse, or from horses to man, there appears to be such a likeness between the maladies that the probabilities are in favour of a similarity in the secondary complications pertaining in both diseases.

The source of infection of the first case of the disease in a stud is often difficult to determine, but influenza is not alone in this respect. The most common source is the introduction of an infected animal. Horses recovered from the disease are also a probable source of infection. How long after recovery the average animal is liable to transmit the disease is unknown, but experience teaches that certain animals may act as reservoirs or carriers, and in support thereof the now well-known case described by Poels is quoted, in which a stallion bought by the Netherlands Government in France transmitted influenza to every mare mated with him for three consecutive seasons.

Probably infection takes place both by inhalation and ingestion, and anything that has been in contact with infected animals may convey the disease to healthy ones. The attendant may do so, and food, litter, clothing, buckets, mangers, etc., are among the most frequent conveyers, as well as stables and railway trucks.

The predisposing causes of the disease are the collection of a number of horses from different localities into one place, such as occurs in the time of war in camps, barracks, ships, etc. In times of peace dealers' stables and other similar stables are common centres for the disease to break out. Anything that lowers the vitality of the animal may be regarded as a predisposing cause, and the crowding of a number of animals together facilitates the spread of the disease. The reason for the increase or the decrease of the virulence in outbreaks is unknown.

Symptoms.—The symptoms presented will vary according to the type of the disease and the presence or absence of complica-

tions. Although the type varies in the outbreaks that occur in different years, it is common to have cases representing all the various types of the disease in each extensive outbreak; in fact, it is not uncommon to have them in a single stud.

Some cases are so mild that it is difficult at times for the practitioner to decide to his own satisfaction whether they are cases of influenza or not. Frequently at the beginning of an outbreak mild cases occur that are followed by a more virulent type of the disease. When this happens, no doubt can be left as to the nature of the disease. In other outbreaks all the cases are of the mild type, and if the nature of the malady has to be decided from clinical observation alone, the most characteristic symptoms of the disease should be borne in mind when doing so. They are: marked injection and often discoloration of the conjunctiva of the eye, accompanied by a high temperature; the great depression of the animal; the œdematous swellings of the skin, particularly of the limbs; and the contagious nature of the complaint.

A mild case may be represented by the following symptoms: The animal is off its food and very dull; there are marked lassitude and a high temperature. Under ordinary febrile treatment and nursing a recovery takes place, without any other symptoms being developed. Next in point of severity, perhaps, comes the type known as catarrhal influenza. In this the foregoing symptoms are present, with the further one of a discharge from the nostrils and a sore throat. This type of the disease is one of degree also. The throat may be considerably swollen, with the nose poked out and the animal having great difficulty in swallowing, particularly water, which runs out of the nostrils nearly as fast as it is taken in by the mouth. The depression is marked, the head hanging, ears drooping, and a general sleepy expression. The animal is disinclined to move, and when it does it is weak and swaying in its gait.

The next type in point of severity is the form known as " pink-eye." Many of the foregoing symptoms are present, and in addition there is a swelling of the eyelids, usually in both eyes, with tears running down the cheeks. The conjunctiva of the eyes is bright red in colour; it may be tinged with yellow. The limbs are swollen and stiff, and movement causes pain.

One of the characteristics of the " pink-eye " type of influenza is the sudden onset of the symptoms and the tendency to attack every other horse in contact.

Taking the various conditions which are regarded as common

complications of influenza, the one most frequently met with is lung trouble. It may take the form of congestion or inflammation of the lungs, and may occur at any stage of the disease. Both lungs are affected, and pleurisy is invariably present. In some cases the affection may be a bronchial one.

The symptoms in such cases are laboured breathing, heaving at the flanks, dilated nostrils, extended head, forelegs wide apart, temperature 106° to 107° F.; the heart's action is weak and flurried. The mortality in this class of cases is high. Stomach and intestinal complications are perhaps present to a varying extent in all cases, but in some instances they are so pronounced that an abdominal form of the disease has been recognised. In some cases the bowels are torpid, and the yellow tinge in the mucous membrane of the eye is a pretty sure indication that the liver and the bile-ducts are affected. At times a mild diarrhœa is present; the fæces are offensive, but if the diarrhœa is not excessive it appears to relieve the patient. The cases more pronounced are those in which colicky pains are present, the animals denoting these by looking round at the flanks, twisting their bodies, etc., more by general uneasiness than in the violent manner of ordinary colic; the bowels are constipated, the dung being hard, dry, and coated with mucus. Owing to the weakness the anus may be dilated, exposing the mucous membrane of the bowel, reddened, dry, with bits of fæces hanging from it. This dilatation is an unfavourable sign. The constipation may be followed by a diarrhœa, and if it is excessive the end usually is not far off.

The nervous symptoms, marked by depression and muscular weakness, that occur in all types of the disease may in certain cases be replaced by more serious nervous complications, of which cerebro-spinal meningitis (inflammation of the membrane covering the brain and spinal cord) is sometimes met with, the symptoms of which are: At first a staggering gait; next the animal gets down, is unable to rise; convulsive struggles follow, the eyes roll, the animal becomes delirious, followed by coma and the end.

In other cases delirium is not present, only coma, or spinal symptoms may be shown by paralysis of the hind-quarters or other sets of muscles, such as the facial, or organs including the penis and the anus.

Heart complications are often the cause of a sudden termination of a case, and the heart, by means of the pulse, is a most important organ to pay attention to in influenza, without going

into finer details of the pulse. The heart is an important organ to watch and note whether the pulse is weak or strong, as upon this the termination of the case to a great extent can be gauged. During convalescence an intermittent or irregular pulse is met with at times, but this usually disappears in due course.

Other complications of influenza are fever in the feet, abortion, strangles, liver or kidney troubles, and thickening of the joints.

Convalescence from severe types of the disease is often a protracted business, and after-effects are in some cases left behind, such as heart or lung trouble, and purpura hæmorrhagica is liable to follow.

Post-mortem Appearances.—The post-mortem appearances will depend upon the type of the case and the nature and extent of the complications. The subcutaneous connective tissue may be infiltrated, the muscles pale in colour and flabby. The blood is the primary seat of the disease, and owing to the toxins produced therein stagnation of the blood is brought about, resulting in congestion of the organs and tissues. There are no typical post-mortem lesions which would enable a definite diagnosis of influenza to be made. The disease can be more certainly diagnosed from the symptoms during life.

Dealing with the various organs of the body more or less in the order in which they are found to show lesions of the disease —the lungs and chest cavity—the windpipe usually contains blood-tinged froth, and patches of inflammation are present on the mucous membrane of the bronchial tubes. Both lungs may be œdematous, much resembling the condition found in horse-sickness, with a patchy pneumonia present which has a tendency to run into gangrene, and abscesses may form in the lungs if the animal lives long enough. This condition is denoted in life by the stench of the animal's breath. Diffuse pleurisy with effusion of blood into the chest is frequently present.

When the digestive tract is involved, inflammation is seen more or less throughout the stomach and intestines. The kidneys are generally congested, and there is a jelly-like substance around them. The heart may be enlarged, inflamed, full of blood, and often covered with blood-spots on its outer surface.

The blood in those cases which have died fairly early in the disease clots very readily. When the disease has lingered on the blood may be thin, and not show a marked tendency to form firm clots.

In the nervous type of the disease effusions will be found in the brain and sometimes in the spine.

Incubation.—Definite knowledge of the period of incubation does not exist. By inoculation the period varies from three and a half to ten days. Some practitioners regard the period as a short one, but perhaps the weight of opinion is in favour of it being from six to eight days. Infection following service by a stallion has been observed to be from six to nine days.

Immunity.—One attack may confer immunity for an indefinite period, but marked variations are met with; two attacks may occur in the same animal within six months of each other.

Treatment.—With the present state of our knowledge, perhaps the best line of treatment that we can adopt is on all fours with that recommended by many medical practitioners in Spanish influenza, and was to this effect: "Go to bed as quickly as possible and stay there." One of the most successful methods of dealing with an outbreak is that of taking the temperature of all the animals regularly, so that all cases may be caught in their earliest stages and the patients, figuratively speaking, "be put to bed," which consists in the best isolation possible from the healthy animals, plenty of fresh air, good easily digested food, and absolute rest. Exercise is deleterious, and any sort of work is fatal to the patient. Whilst the length of the period necessary for convalescence is governed by the type of the disease, it is always advisable to err on the side of making it long rather than short.

Better results will be obtained by a system of good nursing than by medicinal treatment. Bran and linseed mashes help to keep the bowels in order; green food and scalded hay, or a little scalded oat hay, are the best foods for the invalid. Corn is not required. Food should be given in small quantities and fresh, not left in front of the patient.

With regard to medicinal treatment, probably the less this is resorted to the better; attempts should not be made to drench the animal, as the restraint necessary to do so probably does the animal more harm than the drug does good. Of course, it goes without saying that drenching is extremely risky in cases with sore throats. In such cases, and where the discharge from the nose is copious, steaming the patient by pouring boiling water upon some hay in a bucket upon which a teaspoonful of eucalyptus oil has been poured at times apparently gives relief. A light, dry, woollen bandage may also be rolled round the throat.

One line of treatment that can be safely pursued in most cases is Epsom salts This can be given in the drinking water, 4 ounces

to the bucket of water. At the commencement two such doses may be given the first day, and afterwards it can be given in 2, 3, or 4 ounce lots, or it can be dispensed with if not required.

The heart, being greatly depressed, needs stimulation to assist it to relieve the congestion in the various organs, and probably the best agent for the purpose is strychnine injected hypodermically several times daily, or 1 dram of powdered nux vomica mixed with honey or treacle and placed on the tongue answers nearly as well, or carbonate of ammonia may be given as a ball. Quinine, though frequently used, appears to be contra-indicated; it has no action on the virus of the disease, and is a depressant.

In the cases complicated with inflammation of the lungs a mustard plaster frequently affords relief. Take ½ pound of mustard, mix it up with water slightly thinner than it is used on the table, rub this well into the ribs on both sides of the chest for five or ten minutes, and then cover it (plaster) with a sheet of brown paper to keep in the heat.

Different preventive and curative inoculations have been tried, but up to date with only indifferent success, for this disease. It is probable that eventually a successful method of inoculation will be found; in the meantime, reliance has to be placed upon what experience has taught.

SWINE FEVER

Synonyms.—Pig Typhoid, Pneumo-enteritis, Pig Distemper, etc. (American, Hog Cholera).

History.—Probably the disease has existed from the very early times, although there is no definite information of its existence on the continent of Europe until about a century ago. It spread from Europe to Ohio in America in 1832, and it was introduced into Great Britain and Ireland some time prior to 1858. It was discovered in the Cape Colony and Transvaal prior to or in 1903, as in that year, owing to its ravages, it was scheduled as a notifiable (contagious) disease there, also in Rhodesia (1904), but no cases were recorded in the latter country until 1918.

Nature.—Swine fever is a highly contagious eruptive fever peculiar to the pig, and caused by an ultra-visible virus. The manner by which the knowledge that the disease is due to an ultra-visible virus was worked out is most interesting. For many years the cause of the disease was believed to be due to the hog cholera bacillus and organisms which are almost invariably found in animals that have died of the disease. Doubts as to the causal connection of this bacillus were first raised in the United States of America, when an attempt was made to obtain a protective serum by hyperimmunising animals. The serum was at first thought to be very successful, but later experience showed that in the more acute outbreaks the serum was of no use. In the course of his investigations Dr. Schweinitz was led to filter diluted hog cholera blood, and he immediately found that the virus was of the ultra-visible sort; this was in 1903. Other investigators, including Sir John MacFadyean, repeated and elaborated their experiments, and corroborated the finding of the virus being ultra-visible, which is now accepted by all. In 1907 Uhlenhulte, in Berlin, found the hog cholera bacillus present in healthy pigs. It is one of the paratyphoid group of bacilli, and it is now universally accepted that it is a secondary invader, but an important accessory factor, most probably responsible for the characteristic lesions of the disease.

Another most interesting matter in connection with the disease

is that what was known in England years ago as the pneumonic form of swine fever, and of which I had personal experience of several cases, was regarded principally in Germany, but also in Continental countries and America, as a separate disease, and called swine plague, pig pneumonia, swine septicæmia, etc.; but opinion is now coming round to the English view. This form of the disease is ascribed to a small bipolar bacillus that is a normal inhabitant of the healthy pig, and its pathogenic power, like that of the hog cholera bacillus, seems to depend on the invasion of the body by the ultra-visible virus.

Symptoms.—The symptoms vary according as the disease shows itself in the acute, subacute, or chronic form. The various types may exist independently of one another in affected pigs, or the acute may pass into the subacute and the subacute into the chronic.

The Acute Form.—Both experimental and natural infections prove that in very acute cases there may be no characteristic lesions present, but with very few exceptions every pig develops intestinal lesions. Frequently the history of a case is that the owner found the pig dead, and enquiry may elicit the information that the animal fed or appeared to feed well on the previous day, or even at the previous meal. On taking the temperature of the in-contact pigs, if any, it is probable that animals apparently healthy, but really in the early stages of the disease, may be found with temperatures of from 105° to 107° F. If there are acute cases present showing visible symptoms, the temperatures will be found to be high—107° to 110° F.; the animal will be completely prostrated, unable to move, offering little resistance to handling, appearing to be indifferent to everything, and allowing itself to be turned over without a grunt. Discoloration of the ears, abdomen, and thighs may or may not be present. The post-mortem in some such cases reveals no distinct lesions, save perhaps a general inflamed condition of the mucous membrane of the intestine. As a rule, the contents of the bowel are solid and dry. This suddenly fatal form of the disease is generally met with affecting very small pigs or fat stores. Happily for diagnosis there are often other pigs on the premises which appear less acutely affected.

The Subacute Form.—In subacute cases the most typical clinical symptoms are seen, and the period of illness is much longer than in the acute type. The temperature varies, but it is always above the normal (103° F.) unless the pig is nearing death, when it is more often subnormal. The appetite soon fails,

and the animal refuses all food. Frequently there is a desire to vomit, and occasionally vomiting may actually occur, but it is rare. When it does it is usually due to the inflammatory processes being present in the stomach, and later revealed by post-mortem examination. The animal is feverish and shivers from head to foot, the gait is staggering, the steps are short, there is loss of muscular control, the animal sways from side to side and cannot remain standing long at a time. The hind-legs knuckle over at the fetlocks and often cross one another. In some cases it loses the use of its hind-quarters entirely, and drags them along the floor when it moves.

At first the bowels are usually constipated; this is followed by a continuous diarrhœa. The motions are frequent, and vary in colour from black to green, grey, brown, or yellow, possessing an offensive odour, and sticky in nature, only becoming watery in the later stages of the disease. Straining, attended with pain, is frequently present; the motions may be tinged with blood, or clots of blood may be passed. The pig and sty have a peculiar, sour, fœtid odour.

Thirst is a constant and well-marked symptom; the animals can be noticed sucking up any available fluids from the floor of the sty. If the lungs are affected the breathing will be short, quick, and laboured. The skin of the body is at first hot and dry, but during the progress of the disease it is cold and clammy; the extremities are cold and discoloured owing to the weakness of the circulation. A rash may be present or absent. It is, however, very common to find inside the thighs and along the abdomen a pin-head rash upon which black scabs ultimately appear. A short, husky cough is always a constant symptom, but it cannot be considered diagnostic, as pigs cough through other causes. It is of the nature of the animal attempting to clear its throat, and is frequently followed by attempts to vomit. There is a catarrhal discharge from the nostrils or eyes of a sticky nature, the eyelids often being glued together. The animal does not answer the call for food, or, if it does from force of habit, it walks out unsteadily in a dazed and drowsy manner, staggers back again into the sty, and drops down in an exhausted condition. Affected pigs are unable to lie in one position for any length of time. They are restless, constantly raising themselves on their forelegs, and shifting their position from one side to the other as they lie close together for warmth. The movement of one frequently causes some of the others to squeal out with pain, due to the tenderness of the abdomen. The head is carried

low, ears dropped, tail uncurled and limp. A rapid loss of flesh is observed. In suckling sows the milk supply falls off or completely stops. Abortion is a very common occurrence in pregnant animals.

The Chronic Form.—In this form the symptoms are usually not very marked; the animals have a hard, stunted, unthrifty look, and the skin is of a pale ashy colour. It is an important form to recognise, as the animals affected by means of the discharges are capable of transmitting the disease to others, and owing to the indefinite nature of the symptoms in many instances cases are liable to be overlooked. The appetite, though good in some cases, is frequently capricious, and there may be an intermittent (watery) diarrhœa. A husky cough is often present, and the eyelids exhibit a sticky, somewhat swollen appearance. Full-grown pigs may not exhibit any distinct symptoms of the disease, and yet such cases have revealed on post-mortem examination extensive ulceration of the bowels.

Post-mortem Appearances.—In acute cases of the disease the only post-mortem appearance may be that of an intense gastro-enteritis (inflammation of stomach and bowels), but in the majority of cases either ulcerative or diphtheritic lesions are present. The commonest lesion is the so-called swine fever ulcer, which has the following appearance: It is usually approximately circular in shape, and may be of any size up to a two-shilling piece; greyish or at times almost black in colour; flat, often showing concentric rings, and about on a level with or a little above the mucous membrane. The ulcers are in reality circumscribed areas of necrosis, and may be present in scores or only a very few. Their favourite site is in the large intestines. When only a few are present they are frequently at the ileo-cæcal valve—that is, where the small intestine joins the large. They are occasionally found also in the stomach, throat, and on the tongue, and similar lesions are fairly common in the skin of the legs and lips in chronic cases. These lesions may not be found in the bowel of a pig in a state of recovery, or, rather, they may be overlooked. But when ulcers have been present, scars are left that can be detected for a long time afterwards. They are round or oval, and lack the roughness of normal mucous membrane, and are slightly depressed; if recent, they are more vascular.

In the diphtheritic lesion, which is usually preceded by inflammation, a greyish or yellowish exudate is formed on the surface of the mucous membrane of the bowel. It becomes firmly adherent to the mucous membrane, which

undergoes necrosis; therefore, diphtheritic and ulcerative lesions are not sharply defined, and in a good many cases are found together. One seldom gets a continuous diphtheritic film of large extent; it usually occurs as isolated spots, patches, and transverse ridges.

In very acute cases the lymphatic glands throughout almost the whole body may be very much congested. In the less acute cases the intestinal, mesenteric, and inguinal glands are more or less swollen. The spleen and the liver may contain areas of necrosis, and the lungs may also contain similar areas due to the hog cholera bacillus; but in the large majority of the cases in which lung complications are present in swine fever the pneumonia is caused by a bacillus of the fowl cholera type that is called into activity when the pig becomes the subject of swine fever. This pneumonia is characterised by patches of collapse or consolidation, and in my experience of the disease in England it is present in a considerable portion of the cases met with.

Incubation.—The period of incubation is about five to ten days. A rise of temperature is generally found about the fifth day, and visible signs of illness appear about the eighth or tenth day.

Treatment.—There is none. All affected and in-contact pigs are slaughtered.

Certain measures are recommended for preventing the introduction of the disease into premises, and for ridding infected premises of the infection, amongst which the following precautions may be mentioned:

1. Keep animals under good healthy sanitary conditions.

2. Avoid purchasing on markets or from herds unless they are known to be free from the disease—in fact, avoid introducing any animals into the premises when the disease is present in the country. These remarks apply equally to sending pigs to market and bringing them back if unsold, or sending a sow away to a boar and returning her.

3. If the foregoing cannot be avoided, isolate such animals on the arrival at the farm, and have separate attendants and utensils for them. A travelling pig castrator is a possible source of spreading the disease.

With regard to ridding the place of the disease:

1. Once an infected sty has been cleared of its occupants, it should be thoroughly disinfected and left empty for about a month.

2. If there are rats on the premises, they are liable to convey

infective material from one sty to another; therefore efforts should be made to reduce their number.

3. Sows which have recovered from the disease should not be kept for breeding purposes, as there is great possibility of them restarting the disease by infecting their young. All animals that have recovered should be fattened for the butcher.

The disease is not communicable to human beings; therefore the flesh of carcases that set firmly, in animals killed in the earliest stages of the disease, is fit for human consumption. Whether such trade should be allowed is another matter, and would be governed greatly by circumstances.

Preventive Treatment.—Now that it has been discovered that an ultra-visible virus is the cause of the disease, the attempts to confer immunity through the hog cholera bacillus have been dropped, and all thought and attention centred upon finding a protective inoculation with the " filtrable " virus, and it has been found that by hyperimmunising pigs with virulent blood a protective serum can be obtained. This has been used extensively in the United States of America, and is being employed in a very similar manner to what the rinderpest inoculation was employed in South Africa. Two methods are employed:

1. The simultaneous method, which consists in the injection subcutaneously of virulent blood in doses of from 1 to 2 c.c. and serum in doses of from 10 to 60 c.c., according to size of animal, on the opposite side of the body to where the virulent blood is injected. This method conveys an immunity which lasts for several months, and is recommended for herds which have been exposed to infection.

2. The serum alone method, which confers an immunity lasting a few weeks only; but if animals are exposed to infection during this time, an active immunity is conferred.

Considerable success has attended the use of these methods in America, and similar experiments are being carried out with them in Great Britain. It remains to be seen whether these methods under the conditions existing in England will supply an effective and economic manner of dealing with swine fever. The danger of this proving so would appear to lie in the possibility of the recovered or inoculated animal acting as a reservoir or " carrier " of the disease

HORSE-SICKNESS

History.—The disease has been known in South Africa from the earliest days of the European occupation of the subcontinent. It was first recorded in 1719, more than 200 years ago, and since that time it has raged with greater or less severity; for a number of years perhaps but few cases would occur, to be followed by a season of exceptional gravity, in which a great number of animals would die, the countryside in some places being practically denuded of horses. The years 1801 and 1854 are recorded as seasons of great "horse-sickness"; there have been other years nearly, if not equally, as bad, 1914 being a bad year.

The disease appears to be confined to the African continent, and to assume a more virulent or epizootic form in that portion lying near to the Equator. In such localities the disease is present not only every season, but practically at all times of the year. The farther south one goes from the Equator the more seasonal and intermittent the disease becomes, the outbreaks appearing to have a tendency somewhat similar to the weather to run in cycles, with several years of freedom from the disease intervening.

Nature.—The organism or virus causing the disease belongs to the class of ultra-visible, in that it cannot be seen under the highest power of the microscope. Its presence is assumed by reason of the power to transmit the disease by blood inoculation. The virus is a most persistent one, as is proved by the fact that defibrinated blood can be kept for a length of time—up to four years—and still remain infective; neither does decomposition of the blood destroy the organism.

The animals affected are the horse, mule, and donkey, and their susceptibility ranges in the order in which they are placed. It is rare to meet cases of horse-sickness in the donkey. Fatal cases undoubtedly do occur in these animals, but they possess a considerable degree of resistance, which is apparently handed on to their offspring, the mule, which in its turn is more resistant than the horse. Dogs can contract the disease even fatally by eating the flesh of horses that have died from horse-

sickness; the disease can also be artificially transmitted to them by the injection of blood, and in a similar manner it can be conveyed to the goat. A curious fact in connection herewith has been observed: the blood of the dog will infect the horse.

The way the virus gains entry to the body has a considerable effect on the result. Whilst the one-thousandth part of a cubic centimetre or some such infinitesimal dose will produce the disease when injected, it requires 100 to 150 c.c. to do so in the horse when given through the mouth.

FIG. 32.—CASE OF HORSE-SICKNESS (DIKKOP), SHOWING TYPICAL EFFUSION.

The seasons that are bad for horse-sickness are usually those of heavy rainfall, and the time of the season that is worst is not immediately after the first rains have fallen, but after they have been on for two or three months, when the rivers and pools are full of water and the country is in a sodden state; the disease then continues up to the end of the rains and after. Experience has led to the belief that the first severe frost checks it; it is not customary to have cases of horse-sickness a fortnight after the first frost. The foregoing fact, coupled with the further fact that the disease is more prevalent in warm, moist places such as

swamps, river beds, and valleys—in fact, in the places where mosquitoes are to be found in the greatest number—gives colour to the belief that the mosquito is the transmitter of the disease. Whilst no absolute proof has been obtained which will incriminate mosquitoes or a particular mosquito as the transmitter of the disease, experiments have been carried out that fix the responsibility upon some flying insect, and practical experience points to it being a night-flying one.

Different strains of the disease of varying virulence exist in different districts; therefore an animal that has recovered ("salted" to the disease) in one locality may contract the disease again when taken to another locality; such reinfections may end fatally, but as a rule they do not do so if the ordinary precautions taken for this disease are adopted. Again, relapses of the disease, known as "aanmaaning," occur in animals that remain in the same locality in which they "salted" (recovered). This is not invariably so, but a certain number of salted animals show relapses of the disease every season; others, again, only visibly suffer in bad horse-sickness years. In a few the immunity acquired by one attack appears to last them throughout life, provided they are kept under conditions enabling them to get reinfected, as experience points to the probability of animals removed from a horse-sickness to a non-horse-sickness district, or being kept under conditions whereby they cannot become reinfected, losing their immunity to the disease.

Symptoms.—There are three forms in which the disease manifests itself:

1. Dunkop (thin head), or the pulmonary form of the disease.
2. Dikkop (thick head), in which the head swells up greatly.
3. A suppressed form, in which the symptoms are not so well marked and the percentage of recoveries is greater.

Dunkop.—This is the acute form of the disease, and the symptoms frequently develop with such rapidity that some people assert that there are no symptoms visible a few minutes before death. This has not been my experience, although I have seen animals die within the space of an hour from the appearance of symptoms of the disease.

The first symptom usually noticed is that the animal is sluggish in his work, and if he is kept at work the acute symptoms will develop rapidly. The next symptom will be that the animal appears dull and the depressions (supra-orbital fossæ) above the eye will be filled up; later on they will bulge out half as big as a hen's egg. The membrane of the eye is injected and purple in

colour; the breathing is hurried, later on becoming abdominal, showing a well-marked line of heaving in the flank; the pulse is accelerated, and later on can hardly be felt. The animal is very uneasy, constantly shifting, "paddling" with the feet; at times lying down and getting up again quickly. A cough usually develops at this stage, frequently attended at first with a clear serous fluid in a small quantity from the nostrils, the animal then remaining in the standing position until the end, with legs apart, and labouring for breath, or staggering about until it finally falls

FIG. 33.—ŒDEMA FOLLOWING HORSE-SICKNESS. (Photo by Bevan.)

down; the cough persists, and the fluid from the nostrils increases in volume and becomes frothy, or this froth may not appear until after the animal has dropped down dead, when a quantity of it will issue out of the nostrils.

The mortality in this form of the disease may be regarded as 100 per cent. I have seen cases that have had the froth pouring out of the nostrils recover, but they represent less than 1 per cent.

Dikkop.—In this form of the disease the most noticeable symptom is the swelling about the head and neck. The depres-

sion above the eye is usually the first place to become swollen, and it swells out to an enormous extent in some cases, pushing and everting the eyelids, the animal being a distressing sight. The lips and tongue, as well as the whole head, swell up; the swelling also spreads down the neck to the trunk. The other most prominent symptom is the extreme nervous depression. The animal stands in a most listless manner, without indicating

FIG. 34.—HORSE-SICKNESS, SHOWING SWELLING ABOVE THE EYES.

pain or distress. In the fatal cases the horse usually becomes semi-comatose, and stands until it drops dead; in some cases symptoms of the acute form supervene, and froth exudes from the nostrils immediately before or soon after death; while in other cases the nervous depression is not so marked, the animal picking at its food right through, and eventually recovering. A small percentage of recoveries takes place in this form of the disease.

Suppressed Form.—This is the form of the disease that in the milder cases gives rise to arguments as to whether the horse is actually suffering from the disease or not. The symptom that usually incites suspicion is sluggishness, or, in stable language, there is "something wrong," and if this is in the horse-sickness season thoughts fly to this disease at once. Perhaps the only actual symptom that will be seen is that the membrane of the eye is suffused and has the purplish tinge of horse-sickness. In other cases the supra-orbital fossæ will be filled, perhaps bulging to a certain extent; otherwise the animal appears quite normal, eating its food in the ordinary way. In a number of such cases the few symptoms pass off in the course of a week or two; in others, particularly if the animals are worked, well-marked symptoms of dikkop or dunkop may persist for some weeks. I had a mare once that developed this form of the disease, with a swelling over the eye and the conjunctival membrane suffused. After the symptoms disappeared, the slightest exercise would bring them back again. This went on for a matter of two months before I could put her to regular light work; afterwards she developed similar symptoms—dulness and filling in the eye that lasted about a fortnight every season.

In my experience this form conveys a considerable immunity; probably the animals that contract it possess a great amount of natural resistance to the disease, and I have known animals that have recovered from it to be afterwards treated as "salted" and freely exposed to infection, without any untoward results occurring.

It will be observed that I have refrained from mentioning the temperature in describing the symptoms of horse-sickness. Other writers on the disease usually do so, but my experience (a fairly extensive one) is that in naturally acquired cases of the disease the temperature is useless as an aid to diagnosis. In a great number of both severe and mild cases I have found the temperature to be practically normal.

It would appear that the form of horse-sickness developed in the animal depends upon two factors: first, the strain of the disease with which the animal has become infected; and, secondly, the natural individual resistance to the disease of the patient. It is a curious fact that a very large percentage of the horses which are reported to be "salted" are grey in colour. It is customary in localities where the disease is annually present to mate salted males with salted females, as experience has taught that the progeny from such matings survive in greater numbers

than where one or both of the parents are unsalted, presumably for the reason that the greater natural resistance possessed by the parents is transmitted to the offspring.

Symptoms in the Dog.—Bevan describes the symptoms in dogs that had contracted the disease by eating the flesh of a mule as quickened and laboured breathing, high temperature, and death three to eight hours after the onset of the attack. As the disease developed, the jaws became locked tightly and the tongue had a distinct bluish colour. The mouth appeared abnormally dry, and the mucous membranes were anæmic rather than congested.

Post-mortem Appearances.—Frequently there will be a quantity of the yellowish-white froth exuded from the nostrils lying in front of the nose, coupled with the swellings about the eyes, and in the dikkop form of the disease the enlargement of the head and neck presents a typical picture of the first appearance of the animal that has died of horse-sickness. These appearances should not be accepted as absolutely diagnostic of horse-sickness, although they often are. The most constant and reliable lesion is an intense injection of the villous portion of the stomach. There is nothing like the intense dark redness and absence of inflammatory signs in any other disease that I have seen, not even in arsenical poisoning. The intestines, large and small, frequently present a similar streaky, injected appearance. The next most constant symptom is found in the lungs and the heart. The lungs are infiltrated with amber-coloured serosity, and on cutting into them this and froth exudes from the cut surfaces. In cases where the froth has not exuded from the nostrils the smaller and the larger bronchial tubes will frequently be found filled with it. The heart shows hæmorrhage blotchings of a mulberry colour; the heart sac contains a quantity, often a large quantity, of clear fluid, and the chest cavity the same fluid in larger amount. The other organs and portions of the body are usually normal in appearance.

Incubation.—In natural cases my experience leads me to believe this to be between seven and fourteen days, usually between seven and ten.

Treatment—*Curative.*—Practically everything worthy of the name of drug, and many other substances, have been tried in the curative treatment of this disease, and cases have recovered under a variety of treatments; but I think these should be regarded as recoveries in spite of the treatment adopted. The only treatment from which I have seen any good result follow is the

withholding of drinking water from the animal whilst the acute symptoms are present, and afterwards only allowing it in very meagre quantities, such as a pint at a time. I have seen animals live for several days, and show every indication of recovering under this treatment; but upon giving them a bucket of water they have died within an hour.

Preventive.—The first method adopted to prevent this disease was the one of stabling animals at least an hour before sunset, and not allowing them out until two or three hours after sunrise. The origin of this method was founded in the belief that the disease was contracted from the dew on the grass. The dew theory has been disposed of by experiment, and the success which attended the method is now attributed to keeping the animals from night-flying insects.

Another system practised is driving and keeping the animals on the top of the mountains during the horse-sickness season. This also has been found of service.

Another plan is smoking the stables, either by burning wet manure or similar material, or by housing the native servants in the same building, the smoke from their fires permeating through the whole building and making it objectionable to flying insects.

For animals (unsalted) compelled to travel by night, rubbing them over with paraffin and oil, and when they are tied up in the open building a smoky fire to windward of them, has been found helpful. During the Boer War it was observed that horses suffering from mange treated with paraffin, sulphur, and oil, and allowed to run during the night as well as day, escaped the disease.

It has been experimentally proved that in mosquito-proof (screened in with wire gauze) stables horses do not contract the disease in bad horse-sickness localities, although animals standing alongside them in unscreened stables do so. It is a wise precaution to take a horse's temperature daily during the horse-sickness season in areas where the disease is prevalent, and to refrain from working any animal with a high temperature.

With the advent of the cattle-dipping tank the regular weekly dipping of horses in the arsenical cattle-dipping fluid was tried. This apparently acted as a preventive, particularly if oil and paraffin were added and floated on the surface of the fluid in the tank, in mild years of the disease, but in bad seasons the effect was not great.

Preventive Inoculation.—About 1905 Sir Arnold Theiler, who has done a great amount of research work regarding this disease, and to whom much of the knowledge of it is due, brought out a preventive inoculation for mules. This consisted in the simultaneous injection of virus and serum obtained from a hyper-immunised horse. The method has answered admirably, several thousand mules having thus been inoculated, with only a loss of approximately 5 per cent., including those that died during inoculation and others that afterwards became naturally rein-

FIG. 35.—INOCULATING HORSES AGAINST HORSE-SICKNESS.
(Photo by Bevan.)

fected. Later on he extended the method to horses, with good results. The losses were heavier—10 per cent. and over—but still it was a sound commercial undertaking to those living in horse-sickness areas. Unfortunately pernicious anæmia was introduced amongst his laboratory animals, and the horse-sickness inoculation had to be stopped for a time, but has been restarted.

During the last two years Bevan in Rhodesia has introduced a preventive inoculation for horses on somewhat different lines to that of Theiler, in that serum is not employed. Considerable

success has attended his efforts, and his method has promise of eventually proving a great success.

Eradication of the Disease.—A question of absorbing interest is the probability of this disease ever being eradicated from South Africa. History teaches that with advancing civilisation it disappears to a greater or less extent; places that at one time had the disease annually in a virulent form now are free from it, or only get it occasionally. Science has shown that once an animal has recovered it does not carry it in its blood, therefore the question arises, Where and what is the reservoir? When this question has been answered and the transmitter of the disease to horses has been discovered, new views of the possibility of its eradication will present themselves. That such a reservoir is in existence outside of domesticated animals is apparently proved by the fact that horses or mules may contract the disease many miles away from human habitations or domestic animals.

BLUE TONGUE

Synonyms.—Catarrhal Fever or Malarial Catarrhal Fever, the name originally given to it by Hutcheon (Dutch, *Blauwtong* and *Bekziekte*, Mouth Sickness).

Nature.—This disease is frequently likened to horse-sickness, with which it is similar in many respects; for instance, it is a specific, non-contagious disease, readily communicable by inoculation of a small quantity of blood taken from an animal suffering from it. The organism causing it is ultra-visible (unable to be seen under the highest power of the microscope); it will pass through a Berkefeld filter the same as horse-sickness, which is proved by the filtrate conveying it when inoculated. The disease is more prevalent in some seasons, particularly wet ones, than in others; it occurs at the same time in the season, not with the first rains, but when the rainy season has properly set in. It occurs in the same places, such as low-lying land, valleys, etc., and is rarely met with on the mountains. It is checked by similar means—removing the sheep to the highest local altitude, and by shedding and dipping them. It is not contracted during the day, but at night; and similar experiments of placing animals in mosquito-proof stables situated in areas where the disease is prevalent has led to the same conclusion, which is to fix the suspicion on some night-flying insect as the transmitter of it. The disease stops soon after the first frost; the recovered immune animal does not act as a reservoir and a reservoir is suspected to exist in some wild animal. There are other points, but enough has been said to show the similarity between the two diseases. Amongst the points in which it differs from horse-sickness it may be mentioned that the mortality is not so great, as low as 5 to 10 per cent. being a common average, but in bad years and localities the mortality may reach as high as 50 per cent. Drawn blood, although defibrinated or citrated, does not retain its infectivity so long as in the case of horse-sickness, twenty-five to fifty days being the outside limit of it remaining infective; further, attempts

to transmit the disease by ingestion have failed with the majority of experimenters.

Blue tongue is a disease of the sheep alone. The pure-bred merino is the most susceptible of the sheep farmed in South Africa. Cross-breds between this and the Africander, and also the Persian (black-headed), suffer in a considerable degree, but less severely than the pure-bred merino. Pure-bred Africander and Persian sheep possess a greater degree of resistance against contracting it under natural conditions. By inoculation with virulent blood Angora and Boer goats, also calves, harbour the disease in the blood-stream for a limited time—about ten days or slightly longer. Such blood, when inoculated into susceptible sheep drawn within the time stated, will transmit it. The disease is more prevalent in young sheep up to two years old. Lambs whilst suckling rarely contract it. Probably the fact of adult sheep contracting it to a less extent may be accounted for by immunity acquired by them in an earlier stage of life.

History.—It is presumable that this disease has been known at the Cape since its early settlement, but very little notice was taken of it, and its nature was but very little understood until Spreull in the Cape in 1901-1902 carried out a number of valuable and comprehensive experiments in connection therewith. His observations and conclusions were substantiated in most instances in 1904-1905 by Sir Arnold Theiler working upon the disease in the Transvaal.

The disease is well known in most parts of South Africa. In the Union it is more prevalent in the Cape, Natal, and Transvaal than in the Orange Free State and the more western and inland parts of the territory. In Rhodesia the same distribution prevails, the disease being more in evidence in the Umtali, Melsetter, and Inyanga districts and eastern parts of the country than elsewhere.

Symptoms.—The onset of the disease is usually ushered in with a rise of temperature of 104° to 107° F., but, as in the case of horse-sickness, the temperature is to a great extent useless in diagnosis, and there is a marked drop when the visible symptoms appear, which is usually not until about the seventh to the tenth day. But if it is observed that the sheep is looking thin and not feeding as well as usual, an examination of the mouth may reveal a congestion or, on the other hand, a blueness of the mucous membrane.

Often the first symptom noticed is a licking of the lips and

frothing from the mouth. At this stage, if the mouth is examined the mucous membrane will be found to be inflamed and of a purplish-blue colour—hence the name of blue tongue—and the gums and lips swollen. This swelling at times extends to the jaws and the space between the jaw bones, also to the dental pad, causing an abnormal whiteness. In mild cases this condition lasts two or three days, and recovery thereafter takes place rapidly; in other cases the inside of the mouth, including the tongue, becomes raw, and although the animal may have an inclination to feed, gathering and swallowing the food is almost an impossibility. Emaciation is rapid and recovery protracted. Frequently animals that have the mouth lesions mildly have foot lesions severely, and vice versa. The foot lesions are demonstrated by the animal walking lame and tenderly on the heel, like a horse suffering from laminitis. Examination of the feet shows heat and tenderness, and there is a bluish-red line around the coronets, which are swollen and painful on pressure. Whilst the mouth is sore there is great thirst, and if allowed to drink a quantity of water it may set up vomiting, the result in some cases being that a part of the ingesta finds its way into the windpipe and lungs. Emaciation is severe, and this and loss of wool by shedding often proves a greater loss to the owner than actual deaths. Diarrhœa and a twisting of the neck (wryneck) are usually the forerunners of a fatal issue.

Post-mortem Appearances.—As a rule death ensues from debility and extreme emaciation. The lesions in the mouth in most cases have already healed, but the flesh and organs are found pale and bloodless. The post-mortem appearances will to a great extent depend upon the stage of the disease in which the animal succumbed. In those cases occurring at an early stage traces of the ante-mortem symptoms will be found on post-mortem examination. Exceptional instances show inflammation of the true digestive stomach; the other stomachs and bowels may be also congested, but it is only when diarrhœa is present that a general inflammation exists throughout the large and small bowels. The mesenteric glands are usually inflamed and congested. The liver is frequently congested, and, as is to be expected, the gall-bladder is full of bile. The kidneys are also congested and the other organs more or less normal.

On cutting into the chest cavity a little straw-coloured fluid is found in the heart sac. The heart is mostly congested and has blotches (blood-markings) on its inner wall; the blood is unaltered and well clotted. The lungs are always normal, unless

vomiting has occurred and particles of food have gained entrance to the windpipe.

Incubation.—The period of incubation when the disease is naturally contracted is not yet determined. By inoculation the average period is about four days; the fever generally extends from five to seven days; the mouth lesions last from three to five days; the foot lesions appear from fourteen to seventeen days after inoculation. In cases of recovery the whole course of the disease is about twenty-one days.

Treatment.—Many remedies have been tried and recommended as curative treatments, but as the disease is one that runs a more or less definite course, perhaps the less interference and disturbance caused to the animal by medicinal treatment the better. The sick animals should be taken out of the flock and put in sheds, if available, or sheltered spots by themselves, and allowed free access to a lick of common salt. The mouth may be washed out with a weak antiseptic and astringent lotion —chlorate or permanganate of potash, alum, etc. If the nose is swollen and clogged with dry discharge it should be sponged, and other practices of ordinary good nursing pursued as dictated by the symptoms presented, not forgetting to give to the patient soft, succulent, moist food, and water in not too great a quantity at a time.

Preventive Inoculation.—Spreull was the first to show that susceptible sheep could be immunised by inoculation. He found that by hyper-immunisation—that is, repeated inoculations of virulent blood in increasing quantities into recovered animals—the blood-serum of such animals contained strong preventive properties, and by the knowledge thus obtained he was enabled to introduce an inoculation consisting of a simultaneous injection of virulent blood and preventive serum that conveyed an immunity capable of resisting natural infection of the disease. This method in practice was found efficacious; the only objection to it was the cost caused by the hyper-immunisation.

Sir Arnold Theiler in his investigations found that by passage the virus became attenuated—that is, by passing it through susceptible sheep it became weaker and did not kill the inoculated animals. He found that after passage for ten or more times, although there was no mortality, yet the immunity conveyed was equal to that obtained in the animal recovered from a natural attack of the disease. This method, being less expensive, has replaced that of Spreull. It has been

very largely used, and the immunity produced lasts for twelve months or more. It reduces the sheep in condition slightly, but as it is practised in the spring the animals pick up quickly with the green grass. Ewes in lamb should not be inoculated.

H. Watkins-Pitchford, F.R.C.V.S., F.R.S.E., introduced another method of blood inoculation. In his case the blood is attenuated by subjecting it to a temperature of 102° to 117° F.

EPHEMERAL FEVER

Synonyms.—Three-day Sickness, Dengue (from its resemblance to this disease in man).

History.—In a book called "The Heart of Africa," written by Dr. George Schweinfurth about the year 1867, he describes a disease affecting cattle in Central Africa the symptoms of which are identical with "three-day sickness." The disease was described in Egypt by Poit-Bey in 1897, and in some of the outbreaks large numbers of cattle were affected. The writer was the first to record this disease in South Africa, when it occurred near Bulawayo in January, 1907, and by warning the Veterinary Department of the Transvaal prepared them for its advent into that territory. When the disease first appeared in Rhodesia it was as frequently termed "stiff-sickness" as "three-day sickness"; in fact, the former term is still frequently used there, but owing to this being the name in the Union for another disease, the term as applied to ephemeral fever is gradually falling into disuse. Since 1907 the disease has been practically of yearly occurrence in South Africa. In some seasons few if any cases occur; in other years a greater number; but there has never since been a season in which such a large percentage of cattle have been affected. From native reports it would appear that this disease has visited Matabeleland and Khama's country prior to the occupation of the former country by the British. There seems a probability in this, as from history it appears as if the disease had been present in Central and perhaps others parts of Africa for sixty or more years.

Nature.—The disease can be transmitted from an infected to a healthy animal by blood inoculation, and therefore probably it belongs to the group and is caused by an ultra-visible organism. Cattle of all breeds, of both sexes, and irrespective of the condition they are in, are affected. Calves appear to be more resistant to the disease than older animals, and working bullocks and fat cattle suffer more acutely than others. The disease appears during the rainy season, not usually with the first rains, but in the second and third months of the season. Soil apparently

does not affect it, as it occurs equally on different formations. The disease is practically confined to cattle. A few natural cases have been observed in sheep, and the disease, as was demonstrated by Bevan, can be transmitted to these animals by blood inoculation, and a similar temperature reaction followed subcutaneous injection of an emulsion of the spinal cord of an infected ox.

At the end of 1906 the disease was reported to be present in North-Western Rhodesia. In January it was present in the vicinity of Bulawayo, and from there it spread in an easterly and southerly direction. In the east it broke out in the Selukwe, Victoria, and Charter districts; in the south the Matobo, Mangwe, and Plumtree districts. In February, 1907, it was reported in the Vryburg district of Cape Colony, and a little later in the Transkei. Eventually it was reported as being present throughout the whole country. The way it broke out was most puzzling, and caused much speculation as to the means by which it was spread (?). On reference to the map it will be seen that great distances separate most of these places one from the other; therefore the word "spread" seems hardly the correct one—"jumped" would be better, because if the disease did go from one infected centre and infect a new one, it jumped over many miles and left the intervening country free from the disease for a time. The only transmitters that could be thought of at the time capable of such a feat was the locust, swarms of which were in those days in the country. The theory did not find much favour, and as we have had similar outbreaks of the disease since the locust has disappeared, it may be dismissed. A more probable one would be that, owing to climatic or other causes similar to what occur in horse-sickness and heartwater, in certain years midges, mosquitoes, or whatever the transmitter may be, are more prevalent or more virulent; and practically simultaneous outbreaks of the disease a hundred or more miles apart are not due to the spread from an infected centre, but to separate and distinct sporadic outbreaks. When the disease first appeared in South Africa it would have been most alarming if previous warning had not been received of its nature. In some cases 50 per cent. of the cattle went down with the disease and laid unable to move. It required a certain amount of fortitude on the stockowner's part to take things philosophically and believe what he had been told about the recoveries, but there were so many of them that the easiest thing to do was just to go round and look at them.

Symptoms.—These in the majority of cases are so marked that there is little difficulty of diagnosis. Frequently the animal (or animals) will be found down, looking very helpless, and usually in great muscular pain, with swelling of the joints, almost invariably constipated, and the fæces covered with mucus. All cases do not go down, but if an animal lies down it is often unable to rise again. Amongst the first symptoms are dulness, loss of appetite, saliva dribbling from the mouth, suspension of rumination, staring coat, lameness in one or more limbs or a general stiffness of the whole body, giving the animal a wooden appearance; tympanites is frequently present or straining to pass fæces.

In many cases the symptoms disappear nearly as quickly as they developed. In three days (from which the disease takes its name) the majority of animals have recovered; many are sick two days, and a few even less time than that; on the other hand, in rare cases the symptoms persist, and animals are unable to rise for two or three weeks, although the appetite has returned and the bowels are performing their functions normally. Odd cases remain stiff in their gait for a few weeks.

Post-mortem Appearances.—The mortality from the disease is so very low that few opportunities have occurred of observing the lesions, and it is doubtful if any post-mortem appearances can be really regarded as typical of uncomplicated cases. Bevan gives a magenta discoloration of the abomasum similar to that which occurs in horse-sickness, and in another case a congestion of the brain and spinal cord. Sometimes there are degenerative changes in the muscular tissues of the lumbar region.

Incubation.—The period of incubation of the disease under natural conditions is unknown. By blood inoculation it is from two to three days.

Immunity.—It is not customary for an animal to have more than one attack of the disease in the season. Some cases have undoubtedly had two attacks, and it has been claimed that in exceptional cases three attacks have occurred in the same animal. Perhaps it is not unreasonable to suppose that the decreasing percentage of animals affected since the first invasion of the disease may be due to previously acquired immunity.

Treatment.—The best treatment undoubtedly is to leave the animals alone, and not attempt to drench them with any medicines, as, owing to the difficulty of swallowing in nearly all cases by reason of the muscles of the neck and deglutition being affected,

there is great danger of the medicine entering the windpipe, which it does in a number of instances in drenched animals, with fatal results. There is no necessity either for medicinal interference, as the animals recover just as quickly and well without it. Although the mortality is so very low—less than 2 per cent. in uncomplicated cases of the disease—yet the malady gives the stockowners an anxious time, as the animals go down all over the place, and if heavy rains come on during this time and the affected beasts are exposed to the elements, the death-rate will be increased. On the other hand, if the weather is very hot the animals may become very thirsty, and if they are able to move at all, in their struggles to get to water they may injure themselves or fall into holes. There is always a tendency to tympanites, but, as is well known, in all cases, if a beast lies on its side for any length of time it will blow up, and this has to be guarded against. If the place where the animal is lying is hard, it is advisable to turn it from one side to the other occasionally to prevent the formation of bedsores. It is advisable not to lift the animal in an attempt to get it on its feet. In most cases they only flop down again, but if they are sufficiently recovered to stagger about there is the danger of them injuring themselves. It is better to allow them to get up of their own accord, but if a staggering gait persists dram doses of iodide of potassium daily may afford relief. If the animal is unable to rise after a few days, although normal otherwise, a good bed should be put under it, the beast should be frequently turned, and the muscles massaged and a stimulating liniment well rubbed in. If expense is not a hindrance, 1 ounce of gum camphor dissolved in a bottle of cheap dop (Cape brandy) suits well for the purpose, or weak mustard and water with a little turpentine in it ensures a good rubbing and does good. This treatment should be coupled with the administration of dram doses of powdered nux vomica twice daily.

Although the mortality from uncomplicated cases is very low, by the loss of flesh, time with working animals, and milk, the disease is of considerable economic importance. Cows affected go completely off their milk. Some come back to it again, but in lessened quantities; others go more or less completely dry.

FOOT AND MOUTH DISEASE

Synonyms.—Aphthous Fever, Epizootic Aphtha, Eczema Epizootica, Eczema Contagiosa (French, *Fièvre aphtheuse*; German, *Maul- und Klanenseuche*).

History.—The disease has existed in South Africa; it was present in Swaziland in 1892 and in Rhodesia in 1894 to 1895. In fact, about that time it was fairly prevalent throughout South Africa, but it was of a very mild form, and not all the cattle in contact contracted it. Not much in the way of treatment was adopted. Some people rubbed a little salt in the mouth and drove the animals into a clear-running, gritty stream to cleanse the ulcerated feet. Very few animals died. Another outbreak occurred in January, 1904, amongst some imported camels that were stationed at Goromanzi, near Salisbury, but the disease was confined to these animals, and did not spread. South Africa has been free, or at least comparatively free, of the disease since the nineties, but outbreaks have occurred in South-West Africa.

In this connection it is interesting to note the history of the disease in Great Britain. It was first recognised in 1839, and it existed from then until 1886. The prevalence of the disease fluctuated considerably. In 1861 it declined to very narrow limits, but from then on to 1865 it increased. It was scheduled as a notifiable disease in 1869, and in 1878 local authorities were compelled to put the regulations in force. In 1886 only one outbreak occurred, and from that time the disease has reappeared at various times. After skipping a number of years it reappeared again in 1892, 1893, and 1894, and was then absent until 1900, and so it has gone on. It is very interesting to the student to speculate why this disease should spasmodically reappear in Great Britain and why it should have apparently entirely disappeared from South Africa. Climate has probably a great deal to do with it, as the virus of the disease can withstand a considerable amount of cold, but drying up by the rays of the sun or other means quickly destroys it. But probably the chief cause of recurrence in Great Britain lies in its close

proximity to the Continent, where the disease is almost endemic; and as nearly everything that has been in contact with an infected animal may act as a mechanical means of conveying the disease a considerable distance, the outbreaks in Great Britain are attributed to infection brought over from the Continent. It may be brought over unwittingly by a person upon his boots, clothes, or baggage; it may be upon packing in cases or other means; and in Denmark birds have been accused of transporting the infection into the country.

Nature.—Foot and mouth disease is an acute infectious disorder met with chiefly in cattle, sheep, and pigs, although the other domesticated animals are sometimes affected, and occasionally man himself.

The disease is conveyed from an affected animal to others, and the virus, which is ultra-visible, from animals of one species is capable of inducing the malady in animals of a different species. The virus exists especially in the lymph contained in the vesicles, and when the vesicles in the mouth rupture it is present in the saliva. It also occurs in the milk, in the blood up to the time of the formation of the vesicles, and some authorities hold that it exists in all the excreta, in the exhalations from the body, in the expired air, in the tears, and in the nasal and vaginal mucus.

Two forms of the disease are recognised—viz., (1) the simple or benign form; (2) the malignant or septicæmic form. In the former the actual losses by death are comparatively small, being less than 5 per cent.; but the monetary losses from other causes, such as the loss of condition, especially in fat stock, the deleterious effects of the milk on young stock, inflammation of the udder, abortion, complications with the foot troubles by the affected animals shedding their hoofs, and last, but not least, the dislocation to the livestock trade of the infected area or country, all go to make a heavy loss to stockowners, and are a justification for the very strongest measures that are adopted in some countries for the eradication of this disease.

The malignant form of the disease claims a much larger number of victims, and is more severe in its course and after-effects. Up to 50 or more per cent. of the herd may be lost, and as there is no certainty when an outbreak occurs whether the disease is going to assume the simple or malignant form, or whether, if it starts as the former, it may not increase in virulence until it becomes malignant, makes it imperative that all individual outbreaks should be treated with great rigour.

Incubation.—The period of incubation of the disease is about a week. It may develop as early as thirty-six hours, or not show itself for ten or twelve days. The symptoms of the disease in cattle are distinctive, and generally a number are affected at the same time, making the diagnosis easy.

Symptoms.—The first symptoms are a rise in temperature of 103° to 106° F., with accompanying constitutional disturbances: dulness, shivering, staring coat, loss of appetite, stiffness, disinclination to move, loss of milk in cows, grinding of the teeth, etc. These are followed by the typical symptoms of the disease: a dribbling of saliva from the mouth, accompanied by a peculiar smacking of the lips, with the formation of pimples on the inside of the lips and tip of tongue about the size of kaffir-corn seed. They are yellowish-white in colour, and contain a straw-coloured fluid. After two or three days these vesicles rupture, leaving a ragged-looking ulcer, which as a rule heals in about a fortnight.

The foot lesions in cattle may be absent. When they are present, they consist of similar pimples to those seen in the mouth, somewhat smaller in size, and situated between hair and hoof. Of course, manifestations of the foot troubles are given first by the animals being uneasy when standing, shaking the hind-foot, shifting from one foot to another, followed by well-marked lameness, and if the lesions are severe, a separation of the horn from the sensitive portions of the foot may take place. In milch cows similar vesicles are frequently found on the udder and teats.

In the malignant form of the disease the lesions are very variable, and death may occur so suddenly that the disease may be mistaken for anthrax. It can be described as a form of general poisoning of the body. The vesicles may be found also in the throat, gullet, windpipe, and in the stomach. The hoofs may be shed, also the horns. The disease may extend to the lungs, when respiratory symptoms will be shown. If the bowels are affected, a fœtid diarrhœa will be present. The subcutaneous tissues may become infiltrated, and when punctured emit a fœtid odour.

Immunity.—One attack does not confer permanent immunity, but the recovered animals are immune for an indefinite time; probably about six months is the average period.

Treatment.—Precautions are being taken to prevent the importation of the disease into South Africa by prohibiting the importation of animals from countries where the disease exists;

but that precaution has not stopped Great Britain from getting it, and it may not stop South Africa.

Treatment in the benign form of the disease is simple if the animal can be supplied with soft, succulent food, water, shelter, and other comforts; but in this country in a great many instances these facilities would be lacking, and many animals, through their inability to walk to search for food and water, would suffer greatly, and probably succumb from starvation.

Medical treatment is not greatly called for; a little chlorate or nitrate of potash may be given in the drinking water. Borax, chlorate of potash, with glycerine and water, makes a good wash for the mouth. Boric ointment is useful for the udder. The feet should be kept clean, and astringent antiseptic lotions, such as carbolic acid, mixed with sugar of lead and sulphate of zinc, dissolved in water, may be applied. In the malignant form of the disease carbolic acid may be given internally, but it is doubtful if such cases repay the cost of treatment.

VARIOLA, OR POX

Synonyms.—The term " variola " in human medicine signifies small-pox. Pox affects cattle, sheep, horses, goats, pigs, dogs, buffaloes, camels, and monkeys, and is designated according to the animal affected as variola vaccinia (cow-pox), variola ovina (sheep-pox), etc.

The connection between the disease in man and the variolous affections met with in animals is a subject on which marked differences of opinion exist amongst authorities. For instance, the relationship existing between small-pox and cow-pox is an ever-fruitful source for argument that leads to no definite conclusion.

Whether all these diseases should be classed as being due to ultra-visible viruses is also open to discussion, but that variola ovina is due to such a virus is generally accepted; therefore, as all these diseases are very similar except in point of virulence, it may be convenient to discuss them altogether.

Variola in all animals presents a characteristic eruption which passes through certain stages. Although there are certain peculiarities in the eruption and in its location in the various animals, one description of these stages will suffice for all.

1. **The Papular Stage.**—This commences with small red spots resembling flea-bites, which are formed into papules or hard nodules.

2. **The Vesicular Stage.**—The papule becomes a vesicle by the exudation of lymph, which is seen clear and whitish in colour.

3. **The Pustular Stage.**—In this the vesicle becomes a pustule, its contents become turbid and purulent, and its summit assumes a greyish-yellow colour.

4. **The Stage of Desiccation.**—In this the pustule dries up, at first in the form of yellowish scales, which later on become a dark brown crust, which in due course drops off, leaving the well-known " pit " that is well illustrated in a person " pitted " by the small-pox.

In animals, owing to the vesicles and pustules getting ruptured and rubbed off, the process may not always be seen in quite the manner depicted, but usually a recognition of the various stages

can be observed, and in some cases, depending greatly upon the site of the lesions, the process can be watched through the different stages.

The disease occurs in man and sheep in the form of a severe general disease, and with an extensive pock eruption; in most of the other animals the eruptions are almost invariably confined to certain parts of the body, and the disease is usually very mild in character. Therefore it follows that only sheep-pox and also to some extent pox of goats and swine are of any economic importance.

Cow-Pox (Variola Vaccinia).

Cow-pox is a mild affection, usually preceded by no constitutional disturbance, but there may be a slight diminution in the quantity of milk. The eruption appears on the udder and the teats; consequently during the process of milking the vesicles become ruptured, resulting in rather slow-healing sores. The resulting soreness of the udder and teats makes the cow difficult to milk, and this difficulty may remain for some time after recovery; therefore every effort should be made to let the animal pass through the complaint with as little inflammation and pain as possible. It is advisable to keep the udder clean by washing it with weak disinfectant, then drying with a soft towel, afterwards applying some soothing ointment, hazeline ointment being very good for the purpose, and for preventing the sores hardening and forming "cracks," which turn the most docile cow into a kicker. The disease is frequently transmitted by the milkers; therefore this must be guarded against by washing the hands in disinfectant and, if possible, by having a separate milker for infected animals. Fortunately as a rule the disease does not go through all the milking animals; usually it is confined to the heifers and a small number of the cows. Some of the older animals have probably been through the disease before, as is demonstrated by the "pock marks" on their teats or bags.

Bulls, oxen, and young cattle are rarely affected. In calves the eruption is generally situated on the muzzle, lips, and around the nostrils. Milkers frequently contract the disease, the eruption appearing on the hands. And, of course, as is well known, cow-pox is the disease the lymph of which is used as a preventive vaccination against small-pox in man.

Horse-Pox (Variola Equina).

A difference of opinion exists as to the frequency with which the disease occurs, in the sense that it is mixed up with another disease called contagious pustular stomatitis. Some authorities hold that the two diseases are in reality only one disease; another body of authorities hold that they are separate and distinct; but whichever party is correct, so far as South Africa is concerned the disease may be regarded as of rare occurrence. The only occasion on which I have seen it was in the season of 1897 to 1898, when practically every horse in Bulawayo and probably in Rhodesia contracted it.

The typical pock vesicles in the cases that I saw occurred in the mouth, principally on the inner surface of the lips, occasionally on the tongue; they are also said to occur on the nasal mucous membrane, in the heels, and in various parts of the body.

The disease is a comparatively mild affair really, although for a few days the animal appears dull and sleepy and the salivation is profuse. In my experience it usually ran its course in about ten days, and required but very slight treatment. The horse should be put on green food, if obtainable, and other soft foods; the mouth can be washed out with a mild astringent, such as Condy's fluid or chlorate of potash, but I found it gave relief to have a bucket containing Epsom salts, 2 ounces, and ½ ounce of nitre dissolved in a bucketful of cold water constantly beside it, in which the patient could frequently moisten its mouth.

Without venturing to offer an opinion as to whether horse-pox and contagious pustular stomatitis are separate and distinct diseases, the disease that I saw in 1897 to 1898 had every appearance of pox by the manner in which it spread, in its course, and by the symptoms presented.

Sheep-Pox (Variola Ovina)

Synonyms.—Clavelée, Claveau, Picotte (French).

History.—Sheep-pox appeared in Great Britain in 1847, and the outbreak lasted for four years and occasioned great losses. It appeared again in 1862, but was stamped out in four months by the adoption of effective measures. The disease probably originally came from Asia, where it still exists extensively, also in India. In Europe it exists in France and the southern and eastern European countries, as well as in North Africa. It was introduced into German South-West Africa in 1909 with the

importation of Karakul sheep, and it raged with considerable severity and destroyed a great number of sheep.

Nature.—It is an acute and inoculable affection, occurring as an epizootic peculiar to sheep, characterised by a typical variolous eruption on those parts of the body either devoid of wool or scantily covered by it.

Its economic importance is augmented by the occasional high mortality, as well as by the falling out of the wool, which usually entails considerable loss.

The disease is characterised by the intense and diffusible nature of the infection. The virus contained in the dried scabs is believed to be disseminated by the air; therefore it can be carried for considerable distances by the wind, may enter the system by the air breathed through the lungs, or the food may be contaminated by the wind-borne dust.

The infectivity of the affected animal appears to be greater at the termination of the eruption, when the scabs are formed, and the infection may remain in the wool of the living animal from six to eight weeks.

With regard to the susceptibility of the different breeds of sheep, Senior Veterinary Officer Dixon, in commenting on the disease in German South-West Africa in 1910, states: "The loss is found to be very severe, killing 50 to 75 per cent. of Afrikander sheep; amongst merinos the death-rate is 10 to 20 per cent., but the loss of wool is great." Lambs are more susceptible than older animals. Other species of animals and man are not susceptible, except goats, which occasionally become infected.

Symptoms.—The disease is ushered in with a rise in temperature, which may reach 107° F., accompanied by constitutional disturbance of greater or lesser intensity, according to the virulence of the attack; the pulse and respiration are increased in frequency; trembling, arching of the back, eyelids swollen, mucous membranes of the eyes congested, and lachrymation with a serous discharge from the nose are present.

The eruption appears on those parts of the body devoid of, or scantily covered with, wool, and whilst they are typical of the poxes in their various stages, these are severe, and occupy a period of from three to four weeks to complete their course.

Incubation.—The average period is said to be from four to seven days, but it may be as long as twenty days.

Treatment.—Recovery from a natural attack of the disease is said to confer permanent immunity. Preventive inoculation confers immunity for a year or longer.

Vaccination against the disease appears to have a very old origin, and consists of taking the lymph from the vesicles either direct or collecting it in capillary glass tubes and sealing the tube by heating to preserve the lymph for future use. The lymph is used in a similar manner to that adopted in vaccination against small-pox, by scarification of the skin and rubbing the lymph on the scarified surface, the site selected being the under surface of the tail. This method has been found fairly successful, the objection to it being that the vaccinated animals disseminate the disease to neighbouring healthy flocks.

A later and more successful treatment is that obtained by the serum method, which is similar to the simultaneous inoculation for rinderpest. In this case the serum is obtained from hyper-immunised sheep, and is injected into the rump at the same time as virulent lymph is in the ear. Serum alone can also be employed as a curative for infected animals.

Goat-Pox (Variola Caprina).

This disease has been observed principally in Norway. The symptoms and the site of the eruption is very similar to what occurs in the sheep. The duration of the disease is from one to three weeks, and the mortality is not very great.

Swine-Pox (Variola Suilla).

Swine-pox is a very contagious disease, resembling, in its course, complications, and infectivity, sheep-pox. Young pigs are most susceptible, and the mortality may reach as high as 25 per cent. The eruption may occur on any part of the body. The disease is a rare one.

Epithelioma Contagiosum.

Synonyms.—Avian Variola, Fowl-Pox, Chicken-Pox, Epitheliosis, Pigeon-Pox, Canker, Warts.

Nature.—The disease is due to an ultra-visible virus; it is contagious, and usually appears in the autumn, winter, and early spring. It attacks all the feathered inhabitants of the domestic poultry yard as well as most other birds. It frequently co-exists with avian diphtheria. The virus of the disease exists in the blood and in fluids expressed from the sores. The disease is very prevalent in South Africa, and is the cause of a large number of deaths.

Symptoms.—The disease is manifested by the appearance of yellowish-brown, wart-like nodules on those parts of the skin of the head where feathers are few or absent, such as the nostrils, opening of the ears, the comb, wattles, and the eyelids, the last-named being one of the most common sites. The top of the nodule sloughs off, leaving a dark brown ulcerated sore; these sores have a tendency to run together, and parts of the wattles and comb may be thereby destroyed.

Treatment.—All affected birds should be isolated, and the sores dressed with tincture of iodine. It is advisable to examine the healthy birds daily, so as to catch the disease in its earlier stages, and touch the pimples with a match that has been dipped in pure carbolic oil.

INFECTIOUS OR PERNICIOUS ANÆMIA OF HORSES

Under the above heading a disease that is caused by an ultra-visible virus has been studied by various investigators in Europe and other parts of the world. In South Africa its presence has been discovered by Sir Arnold Theiler in connection with the inoculation for horse-sickness, and has caused great difficulty in the hyper-immunisation of animals necessary to supply the serum for the process.

Whatever the nature of the disease may be and the losses it causes in other lands, so far as South Africa is concerned it appears to be more of laboratory importance than it is under natural conditions. At all events, so far as I am aware, no great number of animals have been diagnosed as dying from the disease. Judging from the symptoms described, particularly in the chronic cases, I think I have met with cases of this disease in the past—cases that could neither be classed as biliary fever nor horse-sickness, and in which all the symptoms described occurred, including those of the washed-out appearance of the mucous membrane of the eye, tinged with yellow, the large-sized hæmorrhagic spots in the eye, especially on the membrane nictitans, and debility, etc.

The following is an anæmia in cattle due to some unknown cause:

Bleek-Ziekte (Wasting-away Sickness).

Under the above designation I have frequently come across cases in cattle. The malady is well known to cattle owners, but so far as I am aware it has never been scientifically investi-

gated. The disease is apparently non-contagious, as it is usual to have only a single animal in a herd infected with it. The greatest number that I have ever known to be infected in a herd is five. The only visible symptoms are an unthrifty appearance and a gradual wasting away, accompanied by anæmia. The animal usually lingers on for many months, frequently going through the rainy season, when the grass is plentiful, and dying during the following winter. Cases occasionally recover, and stockowners claim to have effected cures by the administration of a bottle of liquid (warm) fat. The disease attacks both heifers and steers, but is more frequently seen in bullocks two to five years old. Post-mortem examination reveals simply the appearances due to anæmia.

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